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A CONTRIBUTION TO THE PATHOLOGY AND THERAPY OF DYSARTHRIA DUE TO CERTAIN CEREBRAL LESIONS*

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I. Introduction

Interest in the speech and the speech development of spastics is increasing all over the country. Recent publications by different writers show that work is being done to improve the methods which are used in their treatment. Helen R. Fagan (**4,5**), Palmer (**13**), Pusitz (**15**), Robbins (**16**), Carlson (**3**), and Zentay (**22**) emphasize the necessity of utilizing all expedients of modern neurology and logopedia in order to balance the sequelae of injuries to the central nervous system. Very impressive are the two self-descriptive articles written by spastics and published in the *Journal of Speech Disorders*, one anonymously (**1**) and the other by McKibben (**11**).

I suggest the use of the term *dysarthric* rather than *spastic* speech, because the spasms of single muscles or muscle fibres are almost regularly mixed with the paretic stage of others. The name spastic does not denote both kinds of signs, and may even be the cause of neglect of the paretic signs. Their combination complicates the treatment, since even a single muscle may be afflicted in its different fibres both spastically and paretically.

For a preliminary orientation some common signs in certain groups of dysarthrias may be enumerated, but not without the restriction that these signs may be modified in the single case. Such modifications may be due to special anatomical or functional troubles. These various possibilities will be subject to later consideration.

In dysarthrias due to lesions of the pyramidal tract the movements of articulation are sluggish and clumsy, and often hyperrhinolalia is obvious. The total impression is that of blurred speech. The voice is sometimes spastically, sometimes paretically, hoarse and weak, and even aphonia may be present.

In injuries of the frontal brain, or of the pathways leading from the frontal brain to the pons, the impulse of the movements is inhibited. The movements may stop before being completed. Monotony and acceleration are characteristic. Sometimes no

*This article is dedicated to the memory of the late Dr. Max A. Goldstein.

articulation is audible on the end of a phrase, but there is a buzzing, occasionally an interrupted buzzing. Concentration on the pronunciation slows the speech down and makes it more distinct.

Combinations with extra pyramidal signs are not infrequent. Together with Schilder I (**6**) found in such cases a very remarkable deficiency of chest breathing. The pallidum also participates in this picture, producing palilalia and mega- or microphonia. The same is the case in isolated lesions of the striatum pallidum. Palilalia consists in repetitions of syllables, words, and groups of words, especially on the end of a phrase. Megaphonia means shouting, microphonia means a weakness of the voice. Many cases of chorea and athetosis belong to the group of impediments caused by the striatum pallidum, in regard to which Putnam (New York Neuropathological Society, November 11, 1941) certainly is right in saying, as to the question of athetosis and choreo-athetosis, "I find it very difficult to know where one category begins and the other ends."

Another kind of dysarthria is due to injuries of the paths going downwards from the pallidum. Monotony and monorrhythm, signs of fatigue and a blurred articulation are present. Tremor and whispering sometimes complicate the picture. In Wilson's disease the articulation is extremely blurred, and the muscles are of a waxy stiffness.

Cerebellar ataxias are frequently characterized by a rough, compressed monotonous voice. Schilder (**6**) mentioned as typical for cerebellar injuries adiadochokinesis, in which the degree of contraction rapidly diminishes. On the other hand the patients exaggerate the first impulse and interrupt the impulse too early. Scanned speech is another cerebellar symptom. Lesions of the peripheral nerves, the face, the mouth, the pharynx, and the larynx, naturally influence speech and voice.

The most important peripheral dysarthrias are due to lesions of the pneumogastric and the facial nerve. The former is the motor nerve of the soft palate and the motor as well as the sensory nerve of the larynx. Concerning the paralysis and paresis of the laryngeal muscles (paralysis and paresis of the recurrent nerve) I refer the reader to the laryngological literature. (Numbers 2, 3, and 4 of the *Journal of Speech Disorders*, Volume 7, and Number 2 of Volume 8 contain a bibliography of this topic by C. R. Strother.) I want to mention only a single fact which might be of special interest to the neurologist, namely that there is a possibility of discriminating an acute lesion of this nerve from an old one even if the case history is not reliable. Every case of acute paralysis or paresis of the recurrent nerve shows severe disturbances of speech breathing, as has been proved in cases that acquired the lesion during a goiter operation and whose case histories therefore were reliable. Their waste of air and very frequent inhalings are characteristic. If the physician

simply counts the number of inhalations during one phrase, and finds them not too frequent (as compared with his own breathing when he speaks the same phrase) he may be sure that enough time has passed for compensating the breathing trouble. This compensation takes many months. Such a case, therefore, is chronic. The opposite finding of too frequent inhalings is not indicative of acute cases only, because some chronic ones may not have learned yet to compensate their pathological breathing (**7**, 233-235.)

Paralysis or paresis of the palatal motor nerves hinder either entirely or partly the shifting of the velum. Sometimes only one half is damaged, or is more damaged than the other. In such cases the not damaged, or less damaged, part will still rise completely or rise relatively more than the other. Air will enter and pass through the nose also while non-nasal sounds are pronounced, a fact which becomes especially evident to the listener when he hears voiced sounds. All of them show a component which is similar to the physiologically characteristic property of [m], [n] and [ng], namely nasality. This trouble is generally called rhinolalia aperta, but it seems to me that the name "hyperrhinolalia" is more appropriate. The possibility of an "active hyperrhinolalia" will be mentioned later.

Over-contractions of the tensor and levator veli, frequently joined with similar conditions in certain pharyngeal muscles, produce the clinical picture of hyporhinolalia (rhinolalia clausa). Such a state is often of short duration and may also change with the hyperrhinolalia. Choreatic contractions of the velum sometimes are responsible for hyporhinolalia (as well as for the opposite state). The voice sounds as though adenoids were hindering the normal resonance of the nose.

In facial paralysis affecting the third branch it is the lip sounds that suffer. But since articulation between the lower lip and the edges of the upper incisors is a sufficient substitute for bilabial articulation, the functional result of facial paralysis is frequently very insignificant. (If the patient himself has not found this way of compensation, he can be easily taught to use it.) In unilateral paralysis the plosive sounds [b] and [p] may be too "airy" and therefore resemble the fricatives [f], [v] and [w]. (In such cases the patients should be taught to eliminate also the normal half of the upper lip in pronouncing [b] and [p], and to replace the bilabial by the dentolabial articulation. All such attempts are, of course, indicated only if the paralysis cannot be improved or cured.)

I mentioned above the possibility of a combination of spasticity and paretic stages in different muscles, or in different parts of a single muscle. According to investigations on "spasticity" by Phelps (**14**), the first mentioned fact is a general condition in "spastics." He has found that this combination of spastic and paretic conditions is almost always present, and that not every

permanent or temporary over-contraction of the muscles is due to anatomic pathological changes in the centers corresponding to those muscles. This is to say that some muscles, forced to drag along a paretic mate during a certain work, may gradually come into a state of over-contraction. This also holds true so far as the speech muscles are concerned. It can also be easily understood that the muscles finally tire and become functionally paralyzed under the destructive influence of such efforts.

There may exist also different degrees of paresis in different fibres of the same muscle. That becomes obvious if one asks the patient to hold some corks with his lips. I have chosen a series of corks gradually increasing in size by half a millimeter in diameter. Many of the patients with conspicuous signs of damage to the lips could hold one cork, but could not hold the next larger, or smaller, one. That seems to imply that some fibres, especially fibres of the orbicular muscles, are more afflicted than others. In some cases the lips become hard and stiff holding one cork, but become soft and powerless at an effort to hold the next. This observation seems to corroborate the opinion that some fibres may be affected spastically and others paretically.

Frequently we may find out by tactile examination that some muscles are less spastic than others, or that they are even paretic; but it is difficult to judge the condition of the soft palate, which normally rises in order to touch the pharyngeal wall during the pronunciation of all German and English sounds, excepting [m], [n] and [ng]. In the latter sounds, the so-called nasal sounds, the soft palate hangs down and allows the air to enter the nose. A paretic or paralytic state of the soft palate prevents, as has been shown in a preceding paragraph, the closure between the nose and the mouth, thus causing hyperrhinolalia. My former assistant, Weiss, has found that there may also occur an active intention to prevent the palate from rising ("active hyperrhinolalia").

An attempt to analyze dysarthric speech more accurately reveals the following signs: Many dysarthrics have great difficulties in chewing and swallowing and are unable to speak with a morsel of food in the mouth. If the chewing muscles are affected, moving of the jaw will be difficult. A partial or total paralysis of the tongue will increase the difficulties, because the tongue must turn the food in order to mix it with saliva, and to take it to the entrance of the pharynx where the swallowing reflex rises. Patients sometimes drop the food; fluid returns through the nose and choking is a frequent feature, especially in infants. Eating and speaking movements have a common source and that is why every progress in treatment of one of these functions includes progress in the other. When we treat dysarthria we treat chewing and the accompanying tongue movements at the same time. The experience that progress in one of these two functions in dysarthrics influences favorably the other function proves the

theory of the common origin of speaking and chewing (**7**, 179-180).

A general description of the different forms of dysarthric speech is sufficient neither for diagnostic nor for therapeutic purposes. Profound analytic methods, therefore, are strictly indicated.

II. Analytic Investigations on the Partial Functions of the Speech Mechanism

Breathing. While a person is silent the duration of inhalation and of exhalation are almost the same. In singing and speaking, however, as well as in crying, the ratio between inhaling and exhaling changes remarkably. Inhaling is short and deep, exhaling slow. Gutzman (**9**) described "physiologic incongruity" between thoracic and abdominal movements during speech. The abdominal wall sinks before the thorax. Several writers, Schilling (**19**), for instance, describe a lack of physiologic incongruity in some forms of central dysarthria.

Simple optic observations may inform us about some peculiarities of the respiratory movements. Tactile investigations, however, are more reliable. The investigator touches the thorax with one hand and the abdominal wall with the other. Using this simple method I found, as previously mentioned, a lack of thoracic breathing in some cases of dysarthria after encephalitis lethargica; Schilder (**6**) confirmed my finding. The most exact findings have been obtained by pneumography.

Breathing troubles in central dysarthrias may be present in silence or in speaking. During silence breathing is sometimes very shallow, but sometimes it is abnormally deep. Laryngoscopy reveals some of the underlying causes for those facts, as will be shown in the section on "Voice" which follows presently. Congruities between the curve of the thorax and the abdomen may exist. During reading and speaking normal incongruity may be absent permanently or merely occasionally. Inhaling often starts too early, that is to say before the inhaled air is consumed. However, waste of air and consequently a breathy voice are frequently noticeable. The abdominal wall may show movements opposite in phase to the movements of the thoracic wall, which indicates that diaphragmatic respiratory movements are not congruent to those of the ribs. In addition, a quivering of the muscles can be inferred from the appearance of too many peaks contained in the ascending and descending parts of the pneumographic curves. Breathy voice, disturbed modulation and disturbed loudness and pitch are direct consequences of pathological breathing. Not only the vowels but also the consonants will be affected. Waste of air will frequently result in insufficient breath pressure for producing continuous speech. This factor will be especially harmful to explosive sounds but also to all other sounds and will, of course, influence their loudness. Dropping of the jaw as well as ob-

structions of the nose may prevent nose breathing. Our aim to strengthen the muscles which close the mouth will, therefore, be of general value for the system. The different groups of disturbed functions combined with each other explain the immense variety of symptoms briefly mentioned above.

Voice. Attempts to register the movements of the vocal cords with graphic phonetic methods have not been successful as yet. Laryngoscopy is still the most reliable method. The fact that pathological laryngoscopic pictures of dysarthrics do not appear as frequently as would be expected calls for explanation. Everyone not accustomed to a laryngoscopy will move his vocal cords too forcibly. This higher degree of innervation is effective also in cases which normally often fail to give voice. Nevertheless, some pictures valuable for understanding of the pathology have been obtained by laryngoscopy. The movements of the vocal cords during respiration are often abnormal. Normally, exhaling is associated with a triangle-like opening of the glottis with a base of about two to three mm. In our cases this opening is too small in a few instances, but frequently too large owing to flabbiness of the closers or cramps of the openers,or in the first case because of a spasm of the closers and flabbiness of the openers respectively.

Many patients begin an initial vowel explosively, as a result of a too hard closing of the glottis at the beginning of the sound. Physiologists call this hard beginning "hard onset" as compared with a smooth beginning which is called "soft onset."

The voice of a dysarthric can best be judged acoustically. That the pitch is often abnormal and may be either too low or too high, and that there occur deviations from the normal loudness has already been mentioned. Normal inflection is frequently missing and is often exaggerated, especially in states of excitement. West, Kennedy and Carr (**21**) use the word "tenseness" as most descriptive of the quality of these voices.

Systematic examination of the voice is essential. First, one must determine the number of sounds the patient is able to sing. The number of tones, which may be called circumference of the voice, may be very small. The upper resonating cavities, namely, the pharynx, the mouth and the nose influence the pitch and, of course, the timbre. In this connection Dr. D. Weiss' method is very illustrative. A wooden or metallic tube put between one's lips during the singing of a scale will produce a sudden break of voice at a certain pitch. If tubes of different lengths are used, one after the other, the voice will break at different pitches, according to the length of the tube. The tube is nothing but an immobile mouth. This experiment teaches us that the primary tone production can be destroyed or stopped if the tones enter a cavity, the form of which is not fitted to every tone produced. The mouth and the pharyngeal cavity are mobile and adapt themselves instinctively to the momentary pitch. In other words,

these cavities must change their shape for the resonance of different tones. If stiffness or flabbiness of muscles prevents the mouth and pharynx from rapid changes of shape, tones of certain pitches may break or even be destroyed. Therefore, any training of movements of the lips, tongue, palate, and so on will favorably influence articulation as well as voice production. Dysarthrics may benefit from the application of methods used by singing teachers to enlarge the circumference of their students' voices. Those methods serve three common purposes: (a) training of the muscles which stretch and extend the vocal cords, (b) strengthening and regulating exhalation, thus influencing those muscles indirectly, and (c) influencing the function of the vocal cords by improving the shaping of the resonating cavities.

Secondly, we must ascertain whether the patient is able to maintain a certain pitch for a certain time. Different causes may account for a lack of this faculty, such as disturbed respiration, abnormal functioning of the vocal cords or of the resonating cavities, or a combination of these deficiencies.

The so-called "swelling" tones are of the greatest significance in normal speech. A swelling tone is a tone the loudness of which gradually increases or decreases. Many a person with disturbed voice raises or lowers the pitch while increasing or decreasing the loudness. However, the normal swelling tone should not be changed in pitch. Swelling tones are frequently used for emphasis in everyday-life conversation. An unintentional synchronical change in pitch changes the meaning, and must therefore be avoided. Furthermore, dysarthrics frequently lack the ability to jump rapidly from one pitch to another. That the dysarthric cannot sufficiently regulate the loudness of his voice has already been mentioned. The examiner should also ask the patient to give sounds rhythmically in different tempi and at different pitches. A metronome will prove useful for this purpose. Inflection or modulation of the voice is a combination of all the faculties mentioned above and this factor will be very unsatisfactory in spasticity as well as in a condition of flabbiness.

Articulation. An analytic examination of the articulative muscles is somewhat easier than one of the respiratory and laryngeal muscles, because simple optic observation furnishes many important findings. (X-rays, as a rule, are superfluous.) This is probably the reason why some neurological symptoms which indicate the condition of these muscles have been detected. Some troubles in muscular activity may be seen at the first look. If the jaw drops and saliva is moistening the skin we will be fairly well-informed about an affliction in the muscles responsible for the closure of the mouth and for deglutition. In order to localize the site of the defect, we must examine in such a case all the sets of muscles involved in the closing of the mouth. It may happen that the patient can raise and drop the jaw fairly well, but cannot close the lips. On the other hand, he may be

able to close the lips firmly when the examiner helps him to lift the jaw. Sometimes a spasm of the digastric muscle is responsible, because it prevents the jaw from rising. The same result, however, may be the consequence of paralysis of the muscles lifting the jaw (masseter muscle and temporal muscle). In spasticity the masseter reflex may be increased and may be clonic. Sometimes the reflex on one side is stronger than on the other, indicating that there exists a difference in the excitability of the two sides, owing to flaccid paralysis on the one side or spasm on the other, or to unequally strong flabbiness or spasms on both sides. The upward and downward movements of the jaw are especially important for speech. Therefore, we will inform ourselves about those functions by directing the patient to raise and lower the jaw. If he is not able to accomplish these movements, we can use a mirror to help him optically. If necessary, we raise the jaw with our hand in order to feel how much power must be added to the patient's effort, which may indicate, for example, how much resistance arises from the digastric muscle. An analogous investigation must be performed to ascertain the patient's ability to drop the jaw both without and with some resistance produced by the examiner's hand. Furthermore, we must examine the downward drawing and raising of the corners of the mouth as well as of the lips, the drawing sideways and narrowing of the corners of the mouth, and finally the protrusion of the lips. If necessary, we use a mirror and assist the patient with our hand in order to inform ourselves about the degree of impairment.

We test the mobility of the tongue by directing the patient to protrude step by step the tongue toward the teeth, the lips, and out of the mouth cavity, to move the tip gradually to the ridges of the upper and under incisors, to the right and left corner of the mouth, to the upper and lower molar teeth, and to turn it backward to the soft palate. If any deficiency becomes obvious, we use a mirror and assist the patient with a spatula in our hand. To measure the degree of paralysis of the protruding muscles we use a handkerchief for holding the tongue and drawing it slightly forward. Thus, we can find out a great deal about the flabbiness or spastic resistance of the muscles involved. Next we are concerned with the faculty of raising the back of the tongue and its backward drawing. If one side of the tongue is paralyzed, or one side is paralyzed more than the other, the tongue deviates toward the weaker side because the stronger half pushes the weaker half towards the side of the latter.

The jaw, lips and tongue should also be examined with regard to their ability to repeat all these movements. The patient must, for instance, be directed to drop or elevate the jaw several times. Thus, we may find that the movement performed once cannot or can only less accurately be repeated a second or third time. There may be a delay in the repetition so that the normal rate

per second may not be reached. This number is five to seven concerning the jaw. Also the power of the different movements during iteration may deviate from the norm. A metronome is very useful for the examination of iterations.

The pharyngeal reflexes are decreased or absent if the sensibility of the area touched is diminished or destroyed. This may be caused peripherally but it is usually due to a central lesion. Another cause of such disturbed reflexes is palsy of the pharyngeal and palatal muscles. If the soft palate does not rise reflexively because of true (not hysterical) paralysis it will not move in speech either. However, it is possible that the function of this organ may be defective in speech and yet react reflexively without delay.

To inform ourselves about the speech function of the soft palate we look into the opened mouth while the patient says "ah." Abnormalities due to dysarthria which occur during speech are the following: (1) no movement of the soft palate; (2) insufficient movement of the soft palate, that is to say, it does not rise enough to close off the mouth from the nose, thus causing hyperrhinolalia; (3) rising of only half of the soft palate or stronger rising of one half. In the latter case the uvula will be drawn toward the stronger side.

Spacity of the soft palate may produce a picture similar to that of active hyperrhinolalia. Schlesinger (**20**)* described a diminution of hyperrhinolalia in the supine position, where, following the law of gravity, the soft palate sinks down towards the pharyngeal wall. This factor, according to my experience, can also be utilized for differentiation between the paretic and spastic states of the soft palate, because in the patient's supine position the spastic palate resists the influence of gravity, causing the opening between the mouth and the nose to persist.

To inform ourselves about the genuine basis of the speech impediment, all relevant anatomical, neurological, and functional investigations must be carried out. Neither optic nor auditive observations on the lips and the tongue are effective enough to reveal all abnormalities. We, therefore, must use graphic methods for minute investigations of the lips and the tongue. I recommend a very simple and inexpensive apparatus for this purpose. A glass tube adjusted to a small rubber ball, such as that used for instillment into the eyes, is adjusted at its smaller end to a rubber tube. The latter is connected with a writing tambour. The walls of the rubber ball must be thin and easily compressible. If one puts the rubber ball between one's lips and compresses it rhythmically and equally, the writing stylus will lift and drop at regular intervals, and every time rise to about the same height. In dysarthria of the lips we find many irregularities concerning rhythm and strength (height of the elevation). Often the stylus

*Robbins (**17**) described, independently of Schlesinger, the same test.

does not move at all, because the patient's lips lack the strength to compress the rubber ball. The latter should be used in different places on the patient's lips in order to indicate whether or not one part works better than another.

To register the force and rhythmical skill of the tip of the tongue we put the rubber ball behind the upper incisors. If a normal person is ordered to compress it only once with the lips or the tongue, the writer will move gradually until the end of the test. In spastics, however, a jittering of the muscles in question will sometimes show. (The same apparatus can also be used for the treatment. If the patient learns to use his muscles better, he will be able to see the progress in stronger and more regular movements of the stylus, which fact will also stimulate his endeavors. Since the patient is able to observe the movements of the stylus in the air, it is superfluous to use a kymograph for this purpose.)

In examining speech sounds, syllables and words we examine the coordination of all three of these functions: namely, the motility of the articulative muscles, the functioning of the larynx and of respiration. Auditive control alone, or else combined with optic observation, is apt to reveal many deviations from the norm. Vowels, for instance, may involve unsuitable positions of the lips, the tongue, and the soft palate; inability to maintain a certain position for any length of time; sudden interruption of the voice; too high or too low a pitch; trembling of the voice; excessive movements of the Adam's apple. In some cases unsuitable movements will cause the patient to replace the vowel asked for by another. For instance, if he is asked to say [ɪ], there will result [ɑ] instead if the jaw and the back of the tongue drop; [u] will be replaced by [o] if the lips cannot be closed sufficiently.

The pronunciation of all plosives, as well as of the three nasals [m], [n] and [ng], depends upon a closure in one of the three zones of articulation. If this closure is not perfect they will be replaced by friction sounds, provided the patient does not altogether stop his attempt. At the beginning the closure may be strong enough; however, an athetotic movement may interfere with it and open it prematurely. As we know, [b] is a voiced sound and accordingly requires a more complicated co-ordination than [p]. Sometimes the simpler co-ordination replaces the more complicated one and [p] will be given instead of [b], but, strangely enough, also the contrary may happen, especially if a voiced sound precedes the [p]. [m] may be unfavorably influenced by athetotic contractions or spasms of the palate, or by any obstruction of the nose. [f] and [v] are normally spoken with an articulation formed between the under lip and the upper incisors. If the jaw drops a little and the under lip fails to touch the upper incisors, a somewhat larger space between the lip and the teeth will not prevent the pronunciation of these sounds; nor will the acoustic result be greatly changed if the under lip is pursed over

the upper teeth. Friction sounds are generally less vulnerable than plosives. Too large an opening will, of course, detrimentally affect pronunciation. As to the voice, the relationship between [f] and [v] is the same as between [p] and [b], and therefore the pathological variations are analogous. Since the pronunciation of [w], otherwise similar to that of [u], takes more time than [u], it may be impaired by a temporary or permanent inability of the patient to maintain sufficiently long articulation, exhalation or voice production. [ʍ], the voiceless consonant, is sometimes replaced by [w] and vice versa, and, in addition, may suffer from all possibilities enumerated for [w] except that concerning the voice. All friction sounds mentioned above are sometimes spoken like plosives if too strong a muscle action occurs.

[d], [t] and [n], which belong to the second zone of articulation, can be changed in a manner analogous to [b], [p] and [m] which belong to the first zone. Moreover, the tongue may be placed between the teeth. This position in itself does not prevent explosion, but if the closure is too feeble owing to dropping of the jaw, a friction sound similar to one of the *th* sounds will result. If the back of the tongue instead of the tip rises, the sound emitted will depend upon the degree of elevation of the tongue. If the back touches the palate, [g], [k], or [ng] will result; if there is a narrow opening left we shall hear the German "posterior" *ch*.

As a rule, all sounds are influenced by the sounds with which they are connected. A patient who, for example, had in mind a vowel following [d] but replaced the [d] by [g] may need more time to bring the mouth organs into the position necessary to pronounce the vowel than he would have taken if he had really pronounced a [d]. It is even questionable whether the patient can emit at all the vowel at which he had aimed. Our ear is very sensitive to any kind of abnormal pronunciation. The example just given is, of course, one out of thousands. Every faulty pronunciation may open an interminable vicious circle.

[d], [t] and [n] may not be changed remarkably if the blade instead of the tip of the tongue articulates sufficiently with the palate. If this closure, however, is incomplete, the consonants will resemble a hard or soft [s]. With [l] the tongue tip may be pressed too hard against the teeth, or may be put between the teeth. In glossoplegia or under other dysarthric conditions the blade of the tongue may become the articulative part. Dropping of the jaw combined with partial palsy of the tongue may completely interfere with the articulation. In this case, as in many others, a substituting sound may be used if the correct articulation is impossible. Sometimes a "lateral" [l], in which the air escapes merely from one side, or from one side more than from the other, will be emitted; it frequently is combined with lateral sigmatism. In these cases [l] happens to be voiceless, thus resembling the Celtic [l] which is unilateral and voiceless. Because of momentary or permanent failing of the voice during formation

of [l], a voiceless [l] will result even if articulated correctly. [r] is often replaced by [d], [t], voiced [s] and uvular [r]. If the motility of the back of the tongue is affected [g], [k] and the plosive part of [ng] will suffer. The explosion may be too feeble owing to insufficient closure between the back of the tongue and the palate. Here again these sounds may be replaced by friction sounds similar to the German *ch*.

The hard onset very frequently is used instead of the plosives of the third zone of articulation. This abuse, joined with a too intensive closure of the glottis, may provoke nodes of the cords. The habit of exerting the closers of the glottis is transmitted to the muscles narrowing the pharynx and to the articulative muscles. In the pathology of voice it is a general experience that a faulty function of one part of the whole apparatus from the diaphragm up to the articulative muscles is, without exception, transmitted to the other parts. This will be so much the more probable if the central condition stimulates the nerves and muscles to strong contractions and to spasms, as is the case in cerebral "palsy." In this way another vicious circle may be initiated. The abnormal shape of the pharynx and the mouth will detrimentally influence the resonance and diminish the loudness of the voice, thus forcing the patient to apply additional strain, in order to make himself heard.

Some examples for double consonant sounds are combinations of [t] and [ʃ], represented in words such as *child, church,* or *latch* and [d] and [ʒ] represented in words such as *ginger, jelly, edge, soldier,* or *age.* It goes without saying that if one or both of the partial sounds are missing or mispronounced the combination will be affected. However, frequently only the combination is omitted, mispronounced or replaced. A delay in the formation of one sound after the other is enough to mar the pronunciation of a double consonant and can cause a wrong psychological reaction on the part of the patient.

The metronome is an almost essential means for the understanding of dysarthria. We can find out the speed of the beats which the patient is able to follow if we direct him to open and to close the jaw, to spread and to round the lips, to lift and to drop the tip and later the back of the tongue, to push the tongue out of the mouth, and to move the tongue tip to both sides, to speak vowels, isolated consonants, syllables and monosyllabic words with the beats of this instrument. In some cases there is not only an upper but also a lower limit. Some dysarthrics, particularly those affected in the frontal brain or the striatum pallidum, have special difficulties in performing a slow rhythm.

In many cases in which even the other parts of the analytic procedure do not reveal much more than the trained ear of the examiner could hear during a simple conversation, the metronome method helps to detect failures which could not be diagnosed otherwise. One example may illustrate the value of the method

in question. An eight-year-old girl who was born without help in normal condition, birth weight seven pounds, had, after ten weeks, some digestive troubles. She never smiled until she was three years and six months old. Sitting up was accomplished at the age of two years, walking began half a year later, and speaking at five years. She frequently was tired without foregoing strain. During the examination some sounds were missing, namely [g], [k] and [ʃ], all of them at the beginning, in the middle and at the end of words. [g] was replaced by [tʃ], [k] by [t] and [ʃ] by [s]. Her speech was very slow. Every syllable during conversation or repeating took half a second on the average. Protruding and spreading of the lips as well as pushing the tongue out of the mouth could be performed in accordance with 192 on the scale of the metronome; that is, about three times per second. Touching the corners of the mouth with the tongue tip was limited to 112; in other words, the speed of this function was reduced to about two-thirds, measured against that of the other functions. There was also a remarkable retardation in touching the upper teeth with the tongue tip (104) while opening and closing, like spreading and protruding the lips, and also lifting the jaw could be performed at the tempo 192. In accordance with this retardation, syllables with an intitial [l] could not be spoken faster than 104, while for instance [b], [p] and [m] reached 160. That [f] had 132 as the upper limit shows that the slight retraction of the under lip, which must touch the edges of the upper incisors in [f] was delayed, as compared with the simple lifting in [b], [p] and [m]. [t] and [n] were affected analogously to [f].

In order to finish this case history it is necessary to anticipate a few words concerning therapeutic procedures for dysarthrics. By training the defective movements systematically, the tempo of [f], [d], [t] and [n] could be gradually brought to 160, and finally a speeding up of the whole speech up to three syllables, occasionally even four, in a second resulted after three months of treatment three times a week.

The unlimited variations possible and the permanent changes in the speech of a single patient corroborate the viewpoint that the analytic approach is the only one to effect orientation in the maze of the disturbed speech found in dysarthria.

III. Therapy

Though various pictures of dysarthric speech impediments may be presented to the examiner, there is one therapeutic advice which, in my opinion, should never be neglected, namely not to begin with speech exercises before the best possible training of the muscles involved has been achieved. If a speech therapist were to start with speech exercises, thus appealing to all muscles participating in the pronunciation of a single sound, he might increase the unbalanced condition. We therefore must

first take care of the more affected muscles, later of the less affected. This rule applies to the paretic as well as the spastic.

There are some methods which can be used in these conditions for each and every muscle involved in voice and articulation. These methods are: (1) jaw-shaking, (2) the "chewing method," and (3) pushing exercises. Jaw-shaking means a rapid motion of the dropped jaw to both sides. This faculty can be learned from slow to fast only step by step even by non-dysarthrics. It is the pterygoids which perform this movement, but successfully only if the muscles attached to the jaw (mylohoid, geniohyoid, geniohyoglossus, platysma myoides) are not over-contracted. Experience has proved that this condition on the part of all the muscles involved in jaw-shaking works upon the laryngeal muscles, bringing them into a similar state of optimal contraction, and can therefore be used for different functional voice troubles. (Sometimes at the beginning pains may occur in the temporomandibular joint; they are, however, only "gym-pains.")

The chewing method is described in the book, *Practice of Voice and Speech Therapy*. We can chew and speak at the same time without interference of one of these functions by the other. Since the same muscles and nerves are involved in both of them they must be very closely related to each other, if they are not even identical. Association of the idea to speak with the idea that one chews with voice production (as primitive people, in fact, do) has been used successfully for stuttering and many functional voice troubles. Dysarthrics may benefit by it if the chewing aspect is less affected or better trained, which fact sometimes occurs, because the speech aspect frequently is more subjected to psychological complications than the other. The patient to whom the close relationship between the functions has been previously explained is then asked to chew "primitively," namely with opening and closing his mouth, lifting and dropping his tongue, and giving voice at the same time. The result will be a kind of language, provided the patient makes various movements (such as those necessary in chewing food) and does not merely repeat mechanically the same movements. Afterwards he should be directed to use his habitual language, maintaining, however, the idea that also this is nothing but chewing and voice-giving. In this way the patient may become convinced of the fundamental easiness of speaking, and thus eliminate his phobic complications.

Jaw-shaking, as well as the chewing method, is applicable only in cases which suffer from a moderate degree of spasticity in those muscles which are involved in chewing and articulation.

Pushing exercises are effectual almost in any case of paresis, both for the muscles mentioned above and for the muscles of voice production. The patient is advised to clench his fists, to raise them to the nipples, to push them down with great strength, synchronically speaking a vowel, a syllable, shorter and then

longer words, and so on. These exercises are based on the physiologic law that some muscles, though not directly engaged, accompany the work of other muscles with involuntary contractions.

Pushing exercises have been used successfully in hundreds of cases of paralysis of the velum and in cases of functional or organic weakness of the laryngeal muscles. (It may be worth mentioning that they have proved even to be life-savers in severe breathing troubles owing to paresis of the recurrent nerve (7, 237-238.) They can be performed, also, in contrast to the two methods just described above, in severe dysarthric conditions, provided that at least one arm of the patient is freely mobile. Special care of single muscles or single muscle groups, however, will always remain essential, even if one or more of these three kinds of exercises are indicated and applicable. In other words, organic and functional over-contraction, as well as paretic conditions, must be treated also locally. Weakness of the lip muscles, for instance, can be counteracted with bottle corks exercises. If the dysarthric tries, over and over again, to hold those which he failed to hold at the beginning, he may finally succeed. For his first attempts it is advisable that he use a mirror in which he can check the functioning of his lips. Frequently he needs the help of his fingers to assist the weak muscles. This assistance is a frequent feature in our treatment for weakness as well as for choreatic movements and over-contractions. It should be considered a law that the patient should use no more than the absolutely necessary strength for this assistance in order to achieve the greatest possible cooperation of the affected muscles, otherwise they do not recover to such a degree as to do their work without assistance.

Phelps has shown us that one severely damaged muscle may bring some of its mates into a state of hyper- or hypo-activity. Fortunately, the contrary may also occur. An improvement of one or several muscles may favorably influence certain others.

The history of a dysarthric boy, aged 16, may serve as an example. He could hardly close his lips in pronouncing [b], [p] and [m], and showed hyperrhinolalia in syllables with initial lip sounds as well as those with initial tongue sounds. We first treated him with corks, and later, for a few days only, with exercises of lip sounds. Hyperrhinolalia, which had been present previously, was no more noticeable during the exercises. That means that no air passed through the nose while he was speaking lip sounds. However, hyperrhinolalia occurred immediately when he spoke syllables containing sounds of the second zone of articulation, such as [d], [t], [s] and [ʃ], sounds which had not been trained so far. Fig. 1 shows some elevations in the under curve as a result of speaking the syllable *poo* several times into a funnel connected with a tambour. The upper line is almost straight, showing that hardly any air passed through the nose (connected

with the tambour by a glass olive and a rubber tube). Fig. 2 results from an analogous registration of the patient's pronunciation of *too*. Here only a shallow curve is visible owing to the feebleness of air pushes leaving the mouth; the nose curve, however, is very apparent.

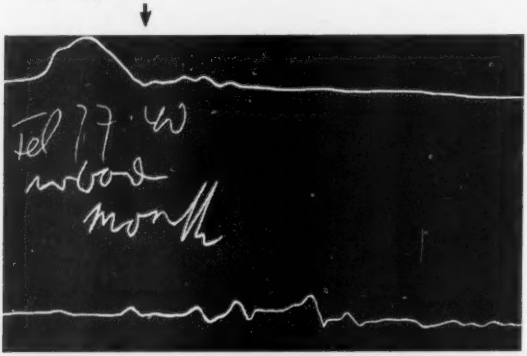

FIG. 1. Record of emission of air from nose (upper line) and mouth (lower line) by a dysarthric boy, aged 16 years, in speaking "poo." Arrow indicates beginning of repeated pronouncing of the syllable.

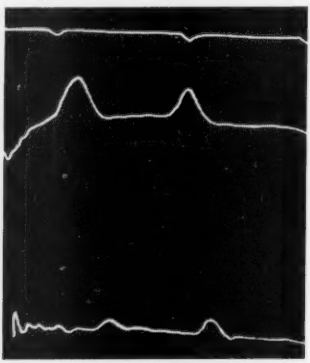

FIG. 2. Record of emission of air from nose (middle line) and mouth (lower line) by the same dysarthric boy represented by Fig. 1, in speaking "too." Arrow indicates beginning of repeated pronouncing of the syllable. Upper line represents time in seconds.

The conclusion to be drawn from these observations seems, as I see it, that systematic training of certain dysarthric muscles is likely to influence favorably other muscles which physiologically collaborate with those first treated—which has also been shown by A. Bullen (2) in non-dysarthric cases.

The whole clinical picture is frequently favorably changed by relaxation training of one muscle or one muscle group. This holds true especially with regard to grimaces. A girl, 14 years old, wrinkled the forehead almost with every word spoken, and nodded her head frequently even when she was silent. Important facts of the case history were breech-delivery, delayed walking, asocial behavior towards other children and pulling of the left

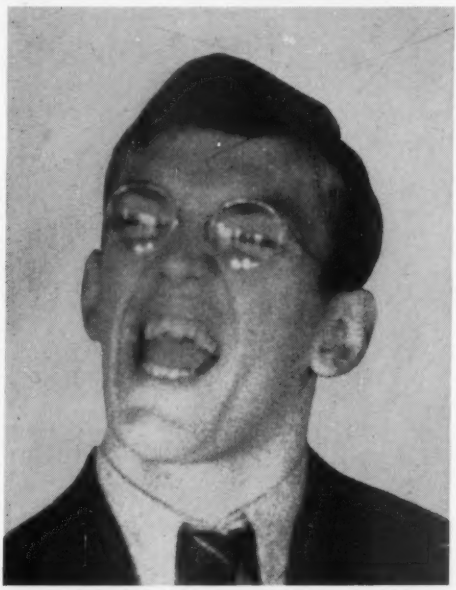

FIG. 3. Production of "ah" by a dysarthric boy before treatment.

leg. The neurological diagnosis was a basal-ganglia disease involving the striatum pallidum. Examination in the speech clinic (six years later) revealed no pulling of the left leg; however, there were athetotic movements in both hands, stronger on the left side than on the right, and ataxy in the left hand which was noted also during the finger-nosetip test. Tactile examination revealed hardness of the bottom of the mouth, while the tongue,

the lips and the cheeks showed no peculiarity. The patient then was taught to relax the muscles of the bottom of the mouth first in rest applying tactile self-examination. Then she was ordered to drop the jaw with the slightest possible muscle contraction. Next came prolonged exhalation through the opened mouth, and lifting and dropping the tip and later on the back of the tongue with dropped jaw. Not before all those coordinations were performed without her wrinkling of the forehead, did we proceed to voice-giving and the articulation of sounds, syllables and words. All of these exercises, again and again, demanded tactile checking of the bottom of the mouth. The treatment has not yet been concluded, it has taken more than a year to get as far as that, since the patient visits the clinic irregularly. She is now able to speak for some minutes without any wrinkling of the forehead. If the wrinkling ever does occur, it is far less obvious than it was at the beginning of the treatment.

FIG. 4. Same boy as shown in Fig. 3, showing immediate effect of moving the tongue toward the stronger side.

Two years ago I developed a method of improving the speech function in cases where the tongue was paralyzed only on one side or on one side more than on the other. The tongue deviates toward the paralyzed side or toward the side where the paralysis is more marked. This phenomenon is based on the fact that the two halves of the genioglossus muscle are orientated with their

fibres toward each other, as has been mentioned before. The stronger part, therefore, pushes the weaker sidewards in such a way that a deviation toward the paralyzed side results. If the tongue tends toward the median line for the purpose of articulation, as it physiologically does in all our sounds, the paralyzed half must struggle against the stronger muscles of the other side. This great effort seems to be the cause of the occurrence of the stronger grimaces. These consist of vivid contractions of the muscles which are innervated by one, two, or all three branches of the facial nerve. An attempt was made to push the tongue of such cases with a laryngoscope or a spatula toward the less

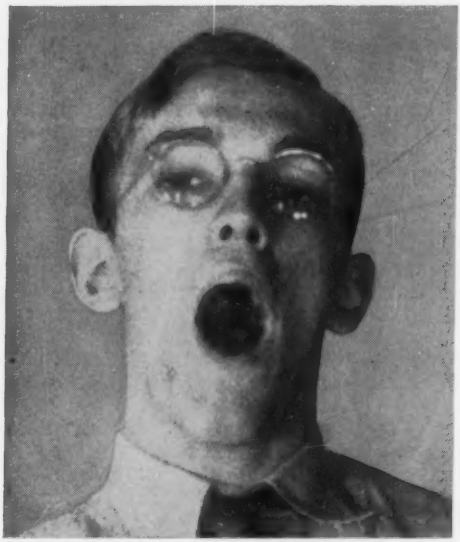

FIG. 5. Production of "ah" by same boy as shown in Fig. 3, after ten days of treatment involving relaxation of the stronger half of the genioglossus, first with mechanical assistance, as shown in Fig. 4, and later without such assistance.

paralyzed side. The tendency is to free the weaker side as far as possible from the pressure of the stronger side. Surprisingly, in some of the cases treated up to now, the grimaces vanished immediately during this help. The patients were told to use this mechanical help during their home exercises of articulation. In a short time these patients learned, by relaxing the stronger half of the genioglossus, to hold the tongue more toward this other, namely the stronger, side, even without the mechanical

help. In this way they finally succeeded in articulating without grimaces and without the aid of the spatula. (See Figures 3, 4 and 5.)

In order to make this article not too long, I must refrain from going into more detail concerning the speech treatment of dysarthrics. A few fundamental rules, however, should be mentioned here. Almost every case needs training of the breathing. The exercises are described in detail in *Practice of Voice and Speech Therapy*. With regard to the teaching of new words, as well as sound correction, no rule can be given as to the order in which sounds should be trained. With some restriction one can follow the rule: From the easier to the more difficult one, the words "easy" and "difficult" being used here only with regard to the special case. It frequently proves easier to begin with syllables than with single sounds. Drills for sounds and phrases can be found in books by Stinchfield-Hawk (**18**), Nemoy-Davis (**12**), Gifford (**8**) and many others. Training of rhythm is almost always essential. The metronome is a good help to begin with.

Initial exercises aiming at relaxation and strengthening of certain muscles should be repeated over and over again. Only experience on the single case can teach us how much time a single training period should take. Generally speaking, it has proved better to have the patient practice several times a day for a few minutes only than to extend the exercises over a longer period. It is necessary, therefore, to teach step by step a member of the family or the nurse to supervise the training. Certain improvements can be attained in almost each and every case, to an extent that will, of course, depend both on the anatomic conditions and on the system of treatment. It should not be forgotten that we have three sources from which to bring about favorable results: (1) reserve material in the central and in the peripheral nervous system; (2) elimination of functional troubles which always accompany the organic and (3) psychotherapeutic help consisting chiefly in encouraging the patient and in focusing his attention upon the speech function.

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PROGRAM COMMITTEE REPORT

As a result of a poll of the Fellowship, the convention of the American Speech Correction Association scheduled to be held in New York City at the Commodore Hotel, December 28-30, 1943, has been cancelled. According to present plans, the convention of the National Association of Teachers of Speech, with which we were scheduled to meet, will proceed as planned. The Program Committee of the American Speech Correction Association is furnishing a general session program for the National Association. A sectional program is also being arranged.

The general session mentioned above will deal with "Speech Correction in War Time." It will include papers on war time speech injuries, general patterns of rehabilitation which are being formulated by national agencies, the rehabilitation activities of the Association, and the place of speech correction in the post-war world.

The Program Committee is hopeful that world conditions will be sufficiently improved so that it will be feasible for the Association to meet in 1944. Preliminary plans for the program of the 1944 meeting are being formulated. These will be announced to the membership in the near future.

RAYMOND CARHART, *Committee Chairman.*

A S C A
News and Announcements

ERNEST H. HENRIKSON, *Editor*
University of Denver

War casualty on the home front is the 1943 convention, which was cancelled by ballot vote of the Fellows of the Association. Thus the sessions of meeting rooms, lobby and coffee shop will have to be postponed until the "business at hand" is disposed of. Pleasant and stimulating as the convention sessions are, there is no indication that the cancellation will cause members to diminish their programs of significant on-the-job activities. Quite the contrary seems to be true.

One very important part of this program which is receiving an increasing rather than a diminishing amount of attention is that which relates directly to the war effort. Special attention of all members is called to the War Notes section edited by Dr. Koepp-Baker. It is a significant record of the part members of the ASCA are playing in the war. More than that, it is suggestive of the increasingly important part which speech correction should have not only in the activities of crisis periods but also in the programs of more "normal" times.

Members of the ASCA can do much to extend the valuable services already being rendered by their association through active cooperation with the defense committees, which were continued by action taken at the Chicago meeting. New readers and old ones who may have forgotten some of the details will want to be reminded that there is a Defense Coordinator, Dr. Herbert Koepp-Baker, and two committees. The Committee on Rehabilitation is composed of Professors Harlan Bloomer, Bryng Bryngelson, Paul Moore, Mack D. Steer, Charles R. Strother, Harold Westlake and Raymond Carhart (Chairman). (Professors Bloomer and Steer are now in service with the United States Navy.) This committee is concerned with problems arising out of war conditions as they create a need for speech rehabilitation among men in the armed forces. (See Dr. Carhart's article in the March issue of the *Journal.*) The Committee on Civilian Defense is made up of Professors James Bender, Harry Heltman, Wendell Johnson, George Kopp, John Snidecor, Ira Wile and Robert West (Chairman).

Since the continued expansion of the Association's value and services can be accelerated through a dynamic increase in the membership of the Association, members are urged to reread Dr. Palmer's article, "American Speech Correction Association Membership Regulations," published in the March issue. Because of the cancellation of the convention, there will

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DIADOCHOKINESIS IN STUTTERERS AND NON-STUTTERERS

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I. INTRODUCTION

In the search for neuro-physiological factors underlying stuttering, a number of studies have been made of the rate and control of movements of various parts of the body musculature.

Anderson (1), in 1923, studied the rate of movement of the foot in tapping and the accuracy of movement of the hand, in a task involving tapping within three circles in a given sequence. He found no significant differences between stutterers and non-stutterers with respect to rate or variability of these movements, with the exception of one group of younger stutterers who differed significantly from their controls in the circle-tapping task. In 1929, West (11) reported research in which significant differences in the combined rates of movement of the jaw and brow were obtained. In 1931, Blackburn (3) reported research which led him to the conclusion that stutterers exceeded non-stutterers in the variability of movement of the diaphragm, tongue, lips, and jaws, but that there was no significant difference in variability of movement in hand-tapping. Westphal (12), in 1933, obtained no significant differences in dynamometer, bean-bag tossing, or Seguin Board tests. Seth (9), in 1934, repeated Blackburn's study and obtained non-significant differences in the variability of the movement of the lips and jaws and a significant difference in mean variability with respect to movements of the diaphragm. Arps (2), in 1934, found stutterers inferior in gymnastic exercises requiring rhythm and coordination. Kiehn (6), in 1935, obtained data indicating a slight inferiority of stutterers to non-stutterers in a tracing test and a test involving carrying a full glass of water. Wulff (13), also in 1935, investigated the rhythm of movement of the lips, jaws, tongue and hands and concluded that, on the average, stutterers are not inferior to non-stutterers in the ability to reproduce given temporal intervals.

Cross (4), in 1936, found no significant differences between stutterers and non-stutterers in unimanual activity as measured by the Koerth Pursuit, Serial Discrimeter, Motor Rhythm, and Tapping tests of the Stanford Motor Skills battery, but did obtain a significant difference in a bimanual task, the Brown Spool-Packing test. She also found non-significant differences between stutterers and right-handed normals in the rate of movement of the left hand, lips, jaws and tongue and significant differences between stutterers and right-handed normals in rate of movement of the right hand and diaphragm.

Hunsley (5), in 1937, reported significant differences between stutterers and non-stutterers in accuracy of reproduction of a rhythm pattern with movements of the lips, jaws, tongue, and diaphragm. Rotter (8), in 1938, found stutterers to be consistently, and in some measures significantly, inferior to non-stutterers in card-sorting and finger-tapping. Spriestersbach (10), in 1940, found no statistically significant differences between stutterers and non-stutterers in rate of movement of the jaws, brow, tongue, and lips.

II. Problem

Inasmuch as these studies have varied considerably with respect to the structures involved, the type of movement required, the apparatus and experimental procedures used, the composition of the experimental groups, the nature of the measures, and the statistical treatment applied, it is difficult to determine the significance of the apparent discrepancies in the results obtained. Because the subject is of considerable importance, it seemed desirable to undertake further research.

Functionally, the structures that have been studied may be classified either as mid-line structures which normally act as a unit, e.g., the lips, jaws, tongue, diaphragm; or as homologous structures which ordinarily act either independently or anti-tropically, e.g., the hands. The movements involved may be classified either as alternating (diadochokinetic) movements, such as tapping, elevation-depression, and adduction-abduction; or as movements involving spatial or temporal discrimination (and possibly other functions). The measures used have been the maximum rate of movement, average rate of movement, variability, and accuracy.

For the purposes of the present study, three mid-line structures, the lips, mandible, and tongue, and one unilateral structure, the right hand, were selected. The hypothesis to be tested was that stutterers do not differ significantly from non-stutterers with respect to the rate of diadochokinetic movements of the lips, mandible, tongue, or finger.

TABLE I. Characteristics of the Experimental (Stuttering) and the Control (Non-Stuttering) Groups.

	Stutterers				Non-Stutterers			
Pair No.	Sex	Age	Dext. Quot.	Rhythm Score	Sex	Age	Dext. Quot.	Rhythm Score
1	f	24-0	81	27	f	20-3	88	27
2	m	18-9	98	23	m	23-1	100	23
3	m	19-8	92	15	m	27-0	100	18
4	m	22-11	100	23	m	27-11	100	24
5	f	18-4	90	20	f	21-5	100	17
6	f	22-0	100	24	f	21-7	95	25
7	m	19-3	90	24	m	23-2	98	26
8	m	25-0	83	25	m	23-4	86	27
9	m	20-8	73	21	m	24-0	78	21
10	m	20-0	83	23	m	23-0	88	22
11	m	27-11	62	16	m	19-8	59	21
12	m	25-4	100	22	m	22-8	96	21
13	m	28-8	100	18	m	31-3	100	19
14	f	31-9	39	22	f	20-8	23	20
15	m	24-0	5	22	m	27-9	0	21
Means		23-0	80	22		24-0	81	22

III. Procedure and Subjects

Subjects

The experimental group consisted of fifteen adult stutterers, eleven male and four female. A control group, obtained from the university population, was matched with the experimental group on the bases of sex, dextrality quotient on the Iowa Laterality Index, and rhythm discrimination on the Seashore test. Table I indicates the closeness of the matching.

Nature of the Movements Studied and of the Apparatus Used

Although evidence obtained from tapping tests indicates that, within limits, diadochokinetic rate is independent of the amplitude of movement, for the purposes of the present experiment it seemed desirable to use a technique which would permit study of the course and amplitude, as well as of the rate, of movement.

In previous studies of tongue movement, various procedures had been used. Blackburn had recorded tongue protrusion by having his subjects place the tongue against a metal plate attached to a tambour diaphragm. Cross' subjects also protruded the tongue against such a tambour diaphragm. Hunsley used a cardiograph tambour with the tongue placed against the diaphragm of the tambour. Wulff had the tongue protruded against an electromagnetic key. Spriestersbach used a small contact plate attached to a rod which closed an electrical circuit when the tongue was protruded. He also recorded tongue-alveolar movement with a contact placed on the alveolar ridge. We have preferred to use the tongue-alveolar movement rather than tongue protrusion since the tongue-alveolar movement occurs more frequently in speech. A pneumatic system was used, consisting of a face mask connected by a rubber tube to a Marey recording tambour. Tongue-alveolar movement was obtained by having the subject repeat the sound *t* with the mask fitted tightly over the mouth. While amplitude of movement of the tongue is not necessarily the only variable affecting the amplitude of successive movements of the recording stylus, the two are sufficiently related for our present purposes.

For recording movement of the jaws, Wulff, West, and Spriestersbach had used levers which closed electrical circuits. Seth had used a scissors-type mechanical lever devised by Gutzmann. Blackburn, Cross, and Hunsley had used a rubber bulb placed between the teeth. We used a rubber bulb fixed between two tongue depressors which were held together by rubber bands placed at either end. Two small tin stops were placed between the depressors. The use of these stops provided better control over the amplitude of movement of the jaw than was possible through the use of a bulb without a stop.

For recording lip movement, Blackburn, Cross, and Hunsley had used lip bulbs, Wulff and Spriestersbach had used lever-type lip keys, and Seth had used Gutzmann's scissors-type mechanical lever. In the present experiment, a small rubber balloon was used. The tip of the balloon was placed between the subject's teeth and the apparatus so arranged that unless the teeth were kept together the balloon would not remain between the lips. This arrangement made it possible to eliminate movement of the jaw while obtaining a sensitive record of the lip movements.

For recording finger-tapping, West, Wulff, and Cross had used lever-type keys. Blackburn had the subject tap on the diaphragm of a modified tambour. Both arm and hand movements were involved in these pro-

cedures. Rotter used a tambour, with a small circular contact plate glued to the diaphragm, and an arm support, with the hand strapped to the support to eliminate other than finger movements. Rotter's technique was used in the present study, with the exception of the taping of the hand to the support, which was found to be unnecessary.

The records were obtained with a Marey recording tambour attached to a constant-speed polygraph.

Experimental Procedure

The first movement was that of the tongue. The subject was asked to make the sound *t* as rapidly as he could. He was then given the mask, instructed to place it tightly against the face, and then asked to make the *t* sound again as rapidly as possible. The polygraph was started and three trials of three seconds each were recorded. The same procedure was used for the other movements.

This procedure does not differ appreciably from that used by other experimenters. Blackburn's subjects and Seth's subjects were instructed to make the movements as regularly as possible, but West, Cross, Spriestersbach, and Rotter instructed their subjects to make the movements as rapidly as possible.

Blackburn recorded four minutes of each movement from stutterers and two minutes from each non-stutterer. Only every fourth second was used in computation of the data. Cross used four trials of five seconds each. Spriestersbach used the records from one trial of three seconds duration. West used the fastest one-second interval in his computations. In the present study, the rate of movement records consisted of three trials of three seconds each. The second of the three trials for each subject was arbitrarily used for scoring and the number of movements in that interval was determined.

The records were measured in units of millimeters. A sheet of onion skin paper was placed directly over the polygraph record and marks were made at the beginning of each movement. The beginning of an upward stroke was considered the beginning of a movement and measurements were made from the beginning of one movement to the beginning of the next.

In order to determine the reliability of measurement, two measurements were made of two patterns from each movement for each of ten records selected at random—a total of eighty patterns. The obtained coefficient of reliability was $.95 \pm .0073$.

IV. Results

The results obtained for the group as a whole, with respect to rate of movement, are given in Table II.

Examination of these data reveals that the stutterers were slightly superior to the non-stutterers in the rate of movement of all the structures studied. In no case, however, does the difference approach statistical significance. In order to determine whether severity of stuttering affected these results, data were computed separately for the severe stutterers and their matched controls. A stutterer was considered "severe" when so rated by not less than three out of four experienced clinicians familiar with his case. These data appear in Table III.

The results for the severe stutterers are substantially the same as those for the entire group. On each movement, the stutterers are consistently, but not significantly, superior to the non-stutterers in rate of movement.

Three previous studies of rate of movement of the tongue have been reported. In order to compare these results with

TABLE II. COMPARISON OF STUTTERERS AND NON-STUTTERERS WITH RESPECT TO RATE OF MOVEMENT OF CERTAIN PERIPHERAL STRUCTURES.

Movement	N	Mean	S.E.m	Diff. (M_{st}-M_{ns})	d.f.*	*t***	Confidence Level***
Tongue							
Stut.	15	7.31	.44				
Non-S.	15	6.53	.37				
				+.78	14	1.25	23%
Jaw							
Stut.	15	4.95	.23				
Non-S.	15	4.76	.30				
				+.19	14	.48	63%
Lips							
Stut.	15	4.55	.19				
Non-S.	15	4.17	.19				
				+.38	14	1.14	27%
Fingers							
Stut.	15	5.06	.42				
Non-S.	15	4.84	.42				
				+.22	14	.69	50%

* d.f. refers to "degrees of freedom," which in this case, since matched groups were used, equals the number of pairs minus one. When the groups are not matched, the d.f. equals $M_{st} + M_{ns} - 2$.

** *t* is a statistic by means of which one can determine the probability of a difference as large as the one obtained occurring by chance. (See Lindquist (7).)

*** "Confidence level," which is obtained from Student's *t* table, may be interpreted to mean that a difference as large as the one obtained in this experiment would be expected to occur *by chance* a given percent of the time.

TABLE III. COMPARISON OF SEVERE STUTTERERS AND NON-STUTTERERS WITH RESPECT TO RATE OF MOVEMENT OF CERTAIN PERIPHERAL STRUCTURES.

Movement	N	Mean	S.E.m	Diff. (M_{st}-M_{ns})	d.f.	*t*	Confidence Level
Tongue							
Stut.	7	7.57	.55				
Non-S.	7	7.23	.31				
				+.34	6	.54	60%
Jaw							
Stut.	7	5.28	.20				
Non-S.	7	4.71	.19				
				+.57	6	2.11	8%
Lips							
Stut.	7	4.66	.17				
Non-S.	7	4.09	.22				
				+.57	6	1.27	25%
Fingers							
Stut.	7	5.57	.38				
Non-S.	7	5.19	.25				
				+.38	6	.73	49%

the results obtained in the present experiment, the necessary statistics were computed and are presented in Table IV.

TABLE IV. COMPARISON OF THE RESULTS OBTAINED BY VARIOUS EXPERIMENTERS WITH RESPECT TO THE DIFFERENCES BETWEEN STUTTERERS AND NON-STUTTERERS IN RATE OF DIADOCHOKINETIC MOVEMENT OF THE TONGUE.

Experimenter	N	Mean	S.E.m	Diff. (M_{st}-M_{ns})	d.f.	t	Confidence Level
Smason and Strother							
Stut.	15	7.31	.44				
Non-S.	15	6.53	.37				
				+.78	14	1.25	23%
Spriestersbach							
Stut.	15	5.20	.28				
Non-S.	15	4.84	.18				
				+.36	28	1.07	29%
Cross*							
Stut.	26	4.31	.25				
Non-S.	42	5.10	.21				
				—.79	(C.R.=2.47)**		1.4%
Blackburn							
Stut.	12	5.04	.30				
Non-S.	13	4.59	.31				
				+.45	23	1.00	33%

* In computing these statistics from Cross' data, results for right and left-handed normals were combined to obtain the mean for the non-stuttering group.

** The C.R. (critical ratio) is a statistic, comparable to t, by means of which one can express the probability that a difference as large as the one obtained might be obtained by chance. The confidence level of a critical ratio is interpreted in the same way as a confidence level for t—in this case, a difference as large as the one obtained would occur by chance 1.4% of the time.

TABLE V. ANALYSIS OF VARIANCE APPLIED TO DATA ON TONGUE RATE OBTAINED BY SPRIESTERSBACH, CROSS, AND BLACKBURN.

	d.f.	Sum of Squares	Variance
Speech	1	1.9785	1.9785*
Experiments	2	1.1987	.5993
Remainder (S x E)	2	10.6250	5.3125

* The *Speech* variance (variance=SD^2) reflects the magnitude of the differences between stutterers and non-stutterers when the data from the three given studies are combined.

The *Experiments* variance reflects the magnitude of the differences between experiments.

The *Remainder* (or, in this case, the *Error*) variance reflects the magnitude of the differences which may be due to chance, to real differences between stutterers and non-stutterers from experiment to experiment or to uncontrolled variables. If the *Remainder* variance exceeds the *Speech* variance, as it does in this case, one is forced to the conclusion that the difference obtained between stutterers and non-stutterers *may* be accounted for by chance. Similarly, if the *Remainder* variance exceeds the *Experiments* variance, as it does here, the differences between experiments *may* be chance differences.

In Cross' study only are the stutterers inferior to the non-stutterers in rate of movement of the tongue. In order to determine whether the pooled results of these studies would reveal significant differences between stutterers and non-stutterers, and also to test the hypothesis that the differences among these experiments might be accounted for by chance, an analysis of variance was applied in the manner suggested by Lindquist (7, 152 ff.), for the analysis of pooled results of duplicated experiments for groups of unequal size. The results of the present experiment were eliminated from this analysis because the close matching of the experimental and control groups in this study precludes its comparison by the variance technique with less closely matched groups[1]. The results of this analysis are given in Table V.

The fact that the Speech variance is less than the Error (Remainder) variance may be interpreted to mean that, for groups similar to those used in these experiments, stutterers do not differ from non-stutterers with respect to rate of diadochokinetic movement of the tongue by an amount greater than might be accounted for by chance. The fact that the Experiments variance also is less than the Error variance may be interpreted to mean that the differences between the results of these experiments are not greater than might be accounted for by chance. The high critical ratio obtained by Cross is, then, to be explained as a chance difference that would be obtained occasionally, although rarely, if a sufficient number of groups of stutterers and non-stutterers were compared.

There have been three previous studies of jaw rate and two studies in which combined jaw-brow rates have been reported. Comparable statistics have been obtained or computed from these studies and are shown in Table VI.

In two of the four studies of jaw rate, the rate of the stutterers exceeds the rate of the non-stutterers; in the other two, the reverse is true. In none of these studies is the obtained difference statistically significant. Although inspection of the previous table indicates that no significant difference would be expected from the pooled results, an analysis of variance was applied with the results shown in Table VII. Neither F is significant at the 5% level. This analysis demonstrates that no significant difference between stutterers and non-stutterers appears when the results of these experiments are pooled.

The two studies in which combined jaw-brow rates were obtained show differences in opposite directions. In West's

[1] One of the basic assumptions involved in analysis of variance is that the variance within a group is the same, except for chance, from group to group. This condition would probably not be met if matched groups were to be compared with unmatched groups.

study, the stutterers are inferior to the non-stutterers by a statistically significant amount; in Spriestersbach's study the stutterers are slightly superior to non-stutterers but the difference is not significant. It is difficult to account for the discrepancy between these results. The experimental procedures were very similar. The only apparent difference would seem to lie in the fact that West's rates were computed from the one-second interval showing the highest rate of movement while Spriestersbach's rates were the mean rates per second for an unselected sample interval of three seconds duration. However, when the rates per second for the jaw movement

TABLE VI. COMPARISON OF STUTTERERS AND NON-STUTTERERS WITH RESPECT TO RATE OF DIADOCHOKINETIC MOVEMENT OF THE JAW, AND FOR JAW AND BROW RATES COMBINED.

Experimenter	N	Mean	S.E.m	Diff. (M_{st}-M_{ns})	d.f.	t	Confidence Level
Smason and Strother							
Stut.	15	4.95	.23				
Non-S.	15	4.76	.30				
				+.19	14	.48	63%
Spriestersbach							
Stut.	15	5.22	.20				
Non-S.	15	5.08	.19				
				+.14	28	.51	61%
Blackburn							
Stut.	8	3.49	.28				
Non-S.	8	4.11	.20				
				—.62	14	1.68	11%
Cross							
Stut.	26	5.14	.18				
Non-S.	42	5.37	.11				
				—.23	(C.R.=1.15)		25%
Combined Jaw and Brow Rates							
West (Males only)							
Stut.	18	7.50	.25				
Non-S.	21	9.26	.38				
				—1.76	37	3.74	0.001%
Spriestersbach (Males only)							
Stut.	15	7.95	.26				
Non-S.	15	7.62	.30				
				+.33	28	.84	40%

only for the *fastest* of 3 three-second trials in the present experiment were computed, the following results were obtained: mean rate for stutterers, 5.06; for non-stutterers, 4.80. The difference (M_{st}—M_{ns}) of .26 yields a t of .74 which, with 14 d.f., is significant only at the 40-50% level. It might still be argued that a significant difference in jaw rate exists for one-second intervals but not for three-second intervals. From the standpoint of neurophysiological theory, such an

TABLE VII. ANALYSIS OF VARIANCE APPLIED TO DATA ON JAW RATES OBTAINED BY SPRIESTERSBACH, BLACKBURN, AND CROSS.

	d.f.	Sum of Squares	Variance
Speech	1	.9739	.9739
Experiments	2	26.8275	13.4137
Remainder (S x E)	2	1.5702	.7851

$$F^* = \frac{\text{Speech}}{\text{S x E}} = 1.24 \qquad \text{At 1 and 2 d.f., P}^{**} > 5\%$$

$$F = \frac{\text{Experiments}}{\text{S x E}} = 17.08 \qquad \text{At 2 and 2 d.f., P} > 5\%$$

* F is a statistic by means of which the significance of a difference between two *variances* may be determined. The formula is:

$$F = \frac{\sigma_1^2}{\sigma_2^2}$$

** The P (or "probability") value is read from an F table (see Lindquist, *op. cit.*, pp. 61-65). In this case, the probability is greater than 5% that an F as large as the one obtained might occur by chance. In other words, in more than five cases out of a hundred, by chance, we would expect the *Speech* variance to exceed the *Remainder* (or, in this case, the *Error*) variance by as much as it does here. Consequently, we cannot be confident that the difference between stutterers and non-stutterers is not due to chance alone.

TABLE VIII. COMPARISON OF THE RESULTS OBTAINED BY VARIOUS EXPERIMENTERS WITH RESPECT TO THE DIFFERENCE BETWEEN STUTTERERS AND NON-STUTTERERS IN RATE OF DIADOCHOKINETIC MOVEMENT OF THE LIPS.

Experimenter	N	Mean	S.E.m	Diff. (M_{st}-M_{ns})	d.f.	t	Confidence Level
Smason and Strother							
Stut.	15	4.55	.19				
Non-S.	15	4.17	.19				
				+.38	14	1.14	27%
Spriestersbach							
Stut.	15	5.51	.22				
Non-S.	15	6.06	.35				
				—.55	28	1.34	19%
Blackburn							
Stut.	8	3.71	.25				
Non S.	8	4.13	.22				
				—.42	14	1.20	26%
Cross							
Stut.	26	5.74	.19				
Non-S.	42	6.00	.15				
				—.26	(C.R.=1.08)		28%

argument is at least questionable, however. It is also conceivable that the difference lies in the brow rates. This possibility should be tested by further research. In view of the fact that four separate studies have failed to reveal significant differences in jaw rate and that one of the two studies of combined jaw-brow rate obtained a non-significant difference, the weight of the evidence would seem to indicate the operation in West's experiment of some factor that did not appear in any of the other studies.

Three previous studies of the rate of movement of the lips have been reported. Table VIII presents a comparison of the results of these experiments.

In all except the present experiment the stutterers prove to be slightly inferior to the non-stutterers in rate of lip movement but in all cases the differences are so small that they might readily be accounted for on the basis of chance. Data for the three comparable experiments were again pooled by analysis of variance, with the results shown in Table IX.

TABLE IX. ANALYSIS OF VARIANCE APPLIED TO THE DATA ON RATE OF MOVEMENT OF THE LIPS OBTAINED BY SPRIESTERSBACH, BLACKBURN, AND CROSS.

	d.f.	Sum of Squares	Variance
Speech	1	3.5938	3.5938
Experiments	2	50.7212	25.3606
Remainder (S x E)	2	.4646	.2323

$$F = \frac{\text{Speech}}{\text{S x E}} = 15.47 \quad \text{At 1 and 2 d.f., } P > 5\%$$

$$F = \frac{\text{Experiments}}{\text{S x E}} = 109.17 \quad \text{At 2 and 2 d.f., } P < 1\%$$

The F for Speech / S x E approaches, but does not reach, the 5% level (the value required at this level, for 1 and 2 d.f., is 18.51), indicating that when the results of these three experiments are combined the differences between stutterers and non-stutterers are not greater than might be attributed to chance. The differences between the experiments, however, as measured by the Experiments / S x E, is significant beyond the 1% level. This would indicate that, although the results of these three experiments are consistent, there are significant differences between the experiments. These differences may be attributed, at least in part, to the relatively low rates of movement obtained in Blackburn's experiment.

There have been four previous studies of tapping rate. Table X presents a comparison of the results of these studies with those obtained in the present experiment.

In all except the present experiment, the stutterers are inferior to the non-stutterers in rate of movement of the finger. In two of the studies the differences are statistically

significant. When the four comparable studies are pooled by analysis of variance, the results shown in Table XI are obtained.

The F for Speech / S x E (Table XI) may be interpreted to mean that, for the groups used in these experiments, a dif-

TABLE X. COMPARISON OF THE RESULTS OBTAINED BY VARIOUS EXPERIMENTERS WITH RESPECT TO THE DIFFERENCE BETWEEN STUTTERERS AND NON-STUTTERERS IN RATE OF DIADOCHOKINETIC MOVEMENT OF THE RIGHT INDEX FINGER (OR RIGHT HAND).

Experimenter	N	Mean	S.E.m	Diff. (M_{st}-M_{ns})	d.f.	t	Confidence Level
Smason and Strother							
Stut.	15	5.06	.42				
Non-S.	15	4.84	.42				
				+.22	14	.69	50%
Blackburn							
Stut.	11	3.68	.24				
Non-S.	10	4.22	.27				
				—.54	19	.62	54%
Rotter							
Stut.	20	5.42*	--				
Non-S.	20	5.71	--				
				—.29	(C.R.=1.53)**		13%
Cross							
Stut.	24	5.44	--				
Non-S.	38	5.87	--				
				—.43	57	2.26	2.8%
West							
Stut.	18	6.93	.19				
Non-S.	21	7.98	.24				
				—1.05	(C.R.=3.50)		0.04%

* Rotter's and Cross' data are stated in terms of the mean number of taps for a 15 second and 40 second interval, respectively. For purposes of the above comparison, and for the analysis of variance, the mean number of taps per second has been computed. The original means, with their respective S.E.m, have been used in determining the t value in the above table.

** This value differs from that reported by Rotter. It was recalculated from Rotter's data. His reported value of 2.72 was apparently in error.

TABLE XI. ANALYSIS OF VARIANCE APPLIED TO DATA ON TAPPING RATE OBTAINED BY BLAKBURN, ROTTER, CROSS, AND WEST.

	d.f.	Sum of Squares	Variance
Speech	1	12.4644	12.4644
Experiments	3	184.7265	61.5755
Remainder (S x E)	3	3.2882	1.0961

$$F=\frac{\text{Speech}}{\text{S x E}}=11.37 \quad \text{At 1 and 3 d.f., } P < 5\%$$

$$F=\frac{\text{Experiments}}{\text{S x E}}=56.18 \quad \text{At 3 and 3 d.f., } P < 1\%$$

ference in tapping rate between stutterers and non-stutterers as great as that obtained would occur by chance less than 5% of the time. This difference remains significant at the 5% level even when West's study is eliminated (because of the much higher means and difference obtained in this study) and Blackburn's, Rotter's, and Cross' results are combined by the Chi-Square method of combining probabilities from independent tests of significance (see Lindquist, *op. cit.*, pp. 46-47). If the probability of a true difference is as great as these two methods of analysis would appear to indicate, why was a difference in the opposite direction obtained in the present experiment? There are two answers to this question. In the first place, these analyses demonstrate only the probability and not the certainty of a true difference. They show that chance might, in 5 cases out of 100, produce groups showing no difference. This may have happened in the present case. In the second place, an explanation may be sought in differences between experiments. That there are significant differences even between the four experiments in which the stutterers were inferior, is demonstrated by the highly significant F obtained in the analysis of variance for Experiments / S x E.

The experiments are known to have differed from one another in a number of ways. Blackburn's instructions differed from those used by the others; Rotter and West used only male subjects; Rotter and Cross computed their means on the basis of longer time intervals. The present experiment differed from all of the others in the fact that the stutterers were closely matched with controls on the basis of sex, age, handedness, and rhythm discrimination. It may be that close matching on one or more of these factors eliminates the differences otherwise found between groups of stutterers and groups of non-stutterers. This possibility must be evaluated by further research.

V. Summary and Conclusions

The present experiment was designed to test the hypothesis that there are no significant differences between stutterers and non-stutterers with respect to rate of diadochokinetic movement of the lips, jaw, tongue, or finger. Fifteen adult stutterers, including both males and females and ranging in age from 18 to 31 years, were closely matched with respect to age, sex, handedness, and rhythm discrimination with a group of fifteen non-stutterers drawn from the college population. Polygraph records of the maximum rate of diadochokinetic movement of the various structures were obtained, and the difference between the two groups in mean rate of movement per second for each of the structures was obtained and evaluated. None of the differences was statistically significant, even

when only severe stutterers were compared with non-stutterers.

Data from several similar experiments, previously reported, were combined and the combined results were evaluated statistically. No significant differences between stutterers and non-stutterers in rate of movement of the tongue, jaw, or lips were revealed by this analysis. A significant difference was found for rate of movement of the right index finger (or right hand) when the results of four independent studies were combined. This finding is not consistent with the results obtained in the present experiment. It is suggested that the explanation of this discrepancy may lie in the fact that, in the present experiment, the stutterers and non-stutterers were very closely matched with respect to sex, age, rhythm discrimination, and handedness.

Further research is needed on different age groups, on the possibility of differences in rate of brow movement, and on the factors related to differences in tapping rate.

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NEWS AND ANNOUNCEMENTS

(Continued from Page 322)

be no direct and personal opportunity to impress members with their responsibility for bringing capable and qualified people into the Association. A review of Dr. Palmer's article should point up this responsibility and should also recall valuable material which members can pass along to interested persons.

Although the ASCA convention has been cancelled, the National Association of Teachers of Speech is scheduled to meet. Many members of the ASCA will attend this convention. Because of the many interests common to members of both associations, this is an opportune time to recall the close ties that exist between the two groups. For example, at the Detroit meeting in December, 1941, it was voted to approve the Educational Exchange Committee, which had previously been approved by the National Association of Teachers of Speech, to function as a joint committee of the two associations. The committee is made up of Professors Wilbur Moore, Elwood Murray, Robert West, Arleigh B. Williamson and D. W. Morris (Chairman). The objective of this committee is to set up and provide for the operation of facilities for the exchange among speech correctionists and teachers of speech of demonstration and instructional materials, such as films, slides, phonograph recordings, etc. The war has imposed drastic limitations on the activities of this committee, but its potential value is such that it was voted at Chicago to continue it. Inquiries or suggestions concerning its work may be addressed to Dr. D. W. Morris, although it is to be remembered that facilities for the actual exchange of materials have not yet been established.

The community of interest that has always existed between the ASCA and the National Association of Teachers of Speech has given rise from time to time to a number of committees and other formally recognized means of cooperation. It is in order to summarize briefly at this time the chief arrangements of this kind that are now in operation. The joint Educational Exchange Committee has already been mentioned. Another means of cooperation between the two associations is provided by the Inter-Organization Convention Committee of the ASCA which at present is composed of President Bryngelson (Chairman), Professor Samuel D. Robbins, (liaison officer for the ASCA in cooperative activities involving our organization and the NATS), Dr. Morris (as Secretary-

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THE ETIOLOGY OF STAMMERING: THE PSYCHOPHYSIOLOGIC FACTS WHICH CONCERN THE PRODUCTION OF SPEECH SOUNDS AND OF STAMMERING*

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In the entire essay which follows I have endeavored to avoid theory, and to state only the provable facts as to the psychophysiologic origin of voice production, and also as to the corresponding facts involved in the production of stammering. Whatever may be the student's conception of the nature of the origin and accomplishment of the psychomuscular acts of stammering, the psychophysiologic facts of the disorder are, I believe, as stated in this essay and any true conception of etiology must, therefore, explain these facts.

The subject of the origin of stammering and of emotion with respect to the disorder must be left by me to future essays.

I. Fundamental Facts Concerning the Protective Bands of the Larynx (Vocal Cords) and Their Activities

The essential and highly active organs within the larynx, the protective bands, or so-called vocal cords, have no subjectively appreciated existence to the child learning to talk, nor to the older child, nor to the adult who has not been informed concerning them. They are neither seen by their possessor, nor perceived understandably by other senses than sight, including the sense of touch and the muscular sense. Their existence and normal as well as abnormal activities are as buried to the mind of their possessor as are the activities of the intestines or the kidneys. Although quite constantly in

* The author has recently published the following four essays on the etiology of stammering. Essay No. 4, (see title below) may be considered as the introductory essay to this new, more detailed and complete study of etiology, begun with the present essay, No. 5. The preceding essays were:

1. Peripheral physical inhibition of speech; an essential phenomenon and an important causal factor of stammering, *Arch. Otolaryngology*, 1930, 12, 769-784.
2. A critical examination of the foundations of the "Recoil of the Vowel" theory of the cause of the impediment of the speech in stammering, *J. Speech Disorders*, 1940, 5, 97-113.
3. The etiology of stammering; an examination into recent studies; with a glance into the future, *Illinois Med. J.*, 1941, 79, 334-341. The above essay appears also in *J. Speech Disorders*, 1941, 6, 1-13.
4. The etiology of stammering: fundamentally a wrong psychophysiologic habit in the control of the vocal cords for the production of an individual speech sound. A beginning presentation, *Illinois Med. J.*, 1942, 81, No. 3. This essay also appears in *J. Speech Disorders*, 1942, 7, 97-105.

action, their movements are known subjectively only by the effects of such movements. This subjective unawareness of the existence of these important organs of the larynx is an important fact. For example, it serves to compel each primary action of such organs to be dependent on fixed involuntary, reflexly controlled, cerebral motor mechanisms. However, certain of these primary animal psychophysiologic functions of the larynx, by virtue of the basal provisions for their action, are also capable of voluntary control.

The two protective bands lie like strong horizontal, movable curtains across the lumen of the larynx. Their anterior ends are attached to the anterior, interior surface of the thyroid cartilage, in close approximation to each other, one on each side of the median line. Posteriorly each protective band is attached to its own arytenoid cartilage, whose whirling movement on the cricoid cartilage serves to open and close each band. The rather simple principle of action of these bands can be readily appreciated by conceiving of their psychophysiologic relation to their cerebral centers.

A fixed cerebral mechanism of control explains why the cricoarytenoideus posticus muscle, which abducts each cord, can act only bilaterally with its fellow of the opposite side. And, likewise, such a mechanism explains why the cricoarytenoideus lateralis muscle, which adducts each cord, can also act only bilaterally with its fellow. Thus the movements of the protective bands are limited (1) to bilateral abduction and (2) to bilateral adduction. The postici muscles, since they abduct the protective bands, are, properly speaking, the muscles of breathing, while the lateralis muscles, since they adduct the protective bands, are the muscles, which, when their action is complete, thus closing the bands, prevent breathing.

Since simple breathing consists of the action of the musculature of the chest wall by which the air is caused to pass in and out of the lungs, breathing as such has no need for an organ like the larynx. And yet, owing to the relation of breathing to other functions, and owing to certain dangers which may menace the lungs and the breathing tube, the protective bands of the larynx are necessary for the conservation and protection of the function of breathing, as well as for ensuring the normal action of other functions which are dependent on breathing, as will be discussed later.

II. The Larynx and the Food Tube

The larynx lies at the top of the trachea, between that organ below and those sections of the upper air passages above, the laryngo-pharynx and the oropharynx, in which the food and air tubes become identical. In swallowing and vomiting the passage of food must take full possession of the laryngo-oropharynx. At that moment the upper air passage must be solely a food tube, so that the passage of food shall not endanger its dropping into the lungs. By intimate cooperation of the body of the larynx with the base of the tongue, with the pharynx and with the mouth of the oesophagus, the oropharynx becomes momentarily solely a food tube, and the lungs are thus protected against the entrance of food particles. During this period of swallowing, or vomiting, the active organs of the larynx, the protective bands, are also doing their part by entering into complete adduction.

The above-mentioned functions of the larynx do not involve the action of the chest and are not concerned with the subject of voice production, or of stammering. They are mentioned here only in order to indicate the full importance of the involvement of the larynx in the basal animal functions.

POSITION OF THE VOCAL CORDS

I.

Vocal cords abducted for breathing.

II.

Vocal cords in a balanced abducted-adducted position for production of the whispered voice.

III.

Vocal cords in a balanced abducted-adducted position for production of the loud type of voice.

IV.

Vocal cords in a state of complete adduction for prevention of breathing,—their position in the act of "blocking."

Passage from position I to position IV constitutes the act of adduction.

Passage from position IV to position I constitutes the act of abduction.

This cut is reprinted from Essay 4.

III. The Voluntary Chest-Laryngeal Animal Functions

The following described animal chest-protective band functions are directly concerned with stammering, and indeed constitute the necessary bases on which stammering becomes founded.

In the performance of the following animal functions the laryngeal box remains independently separated from other organs. Also, each manner of action, being dependent on a fixed cerebral function of control, is associated with the cooperation of the breathing muscles of the chest. In these functions the larynx acts solely through the movements of its own protective bands. The soft palate may, or may not, also enter cooperatively into a particular manner of activity. These functions are coughing and sneezing, with cooperation of the soft palate; and compression of the abdomen, without such cooperation. Each of these chest-protective band functions requires complete adduction of the protective bands, and,

except for sneezing, which is solely an involuntary act, may be either voluntarily or involuntarily produced.

The complete function of breathing involves not only the expansion and compression of the chest wall to produce the inward and outward movements of the breathed air, but it involves also the adjustment of the protective bands for breathing, as well as for meeting the constantly recurring demand for the execution of the functions just mentioned.

Thus the muscles of the larynx are ever in a state of tonicity, instantly prepared to enter on required readjustments. This state of tonic preparedness of the laryngeal muscles is evidenced to the eye as one observes a little start of the protective bands at the beginning of each act of breathing.

Suppose, at any moment, a sensation at some point in the lungs or in the upper air passages demands the incitation of cough for the elimination of the physical cause for such sensation, then, either incited by reflex or by voluntary cerebral action, the following protective activities instantly take place, one following its predecessor in rapid succession:

1. Chest expansion, coupled with wide abduction of the protective bands, by which activities a quick, strong inward breath is taken;
2. Complete adduction of the protective bands, instantly followed by strong compressive chest action, thus momentarily compressing the air of the lungs against the completely closed protective bands;
3. Quick abduction of the protective bands, coupled with stronger compression of the chest, by which the air just held under pressure beneath and against the completely closed protective bands, is caused to burst out between them. In cooperation the soft palate is closed, thus directing the compressed air outwards through the mouth.

As just mentioned, these activities are those of the function of coughing, designed to remove undesirable substances from the lungs and the upper air passages. Together they are produced by a fixed cerebro-chest-protective band neuromuscular mechanism, set into action either by reflex or voluntary excitation.

Moreover, these combinations of action of chest-vocal cord muscles required for the production of cough are also susceptible to *individual* voluntary control, as follows: (1) compression of the chest wall with abduction of the protective bands (for outward breathing); (2) expansion of the chest wall with abduction of the protective bands (for inward breathing); (3) compression of the chest wall with adduction of the protective bands for prevention of outward breathing; and (4) expansion of the chest wall with adduction of the protective bands for prevention of inward breathing.

Except for cough the only other voluntary-involuntary psychophysiologic act of chest compression combined with complete adduction of the protective bands of the larynx, is that of compression of the abdomen (for the expulsion of the faeces, and of the infant in childbirth). Since, by reason of the muscular systems involved in this act, such abdominal compression is necessarily accompanied also by chest compression, the prevention of the escape of air from the lungs is essential for its complete success. This act in actual use is either involuntarily or voluntarily executed.

IV. Vocal Cord Adduction

The normal acts of protective band bilateral abduction, and adduction, are each determined by a fixed involuntary

cerebro-chest-protective band mechanism, which is also capable of voluntary control. In performing voluntary abduction of the vocal cords for breathing one has only to conceive of chest compression, or expansion, with the thought purpose of breathing. The required fixed psychophysiologic mechanism is always ready to respond to such mental imagery. The frequency with which voluntary breathing is carried out as an independent act renders its achievement easy and natural. This we all can appreciate.

Also the conception of chest compression, or expansion, accompanied by the thought purpose that breathed air shall *not* pass through the air tube results in vocal cord adduction. The response to such mental imagery for the production of vocal cord adduction is just as natural and easy as the response of its corresponding mechanism to the mental imagery of breathing, *if one has had equivalent experience.* In coughing or in compression of the abdomen this act is carried out with the utmost ease and naturalness; but these acts are practically never executed with clear realization of their involvement in vocal cord adduction. We must remember that one cannot subjectively appreciate vocal cord existence, or movement.

We must look upon voluntary adduction of the protective bands of the larynx, *made as an independent act,* as almost an unknown normal activity. I doubt whether any otolaryngolist has ever seen the vocal cords in a state of complete adduction. The reason is that a close understanding especially of the less well-known activities of the vocal cords is not called for by his work with respect to them. He is not concerned with educating his patients to exhibit their vocal cords in a state of complete adduction. And yet, in spite of the obscurity of this little known voluntary independent act of laryngeal protective band adduction, if any individual chooses (without thought of cough or of compression of the abdomen) to compress or to expand the chest wall while carrying the thought purpose at the same time that no air shall pass through the air passages, the protective bands enter instantly into complete adduction; and if the individual repeats this act a sufficient number of times (apart from coughing or compression of the abdomen) it becomes just as easy to produce as does the act of abduction of the protective bands for breathing. This statement can be verified by any reader of this essay. I have proved it on many—fifty or more—different individuals. Complete independent voluntary adduction of the protective bands of the larynx for stoppage of breathing, then, is just as natural as is their voluntary abduction for breathing, and is just as readily and easily superinduced as is the act of abduction of the protective bands for breathing, *if it be repeated a corresponding number of times.* This, as we shall see, is accomplished thoroughly by the stammerer.

V. The One Secondary Voluntary Activity of the Chest-Vocal Cord Systems of Movement Is Limited to Sound Production

I have already called attention to the fact that the vocal cords possess but two primary animal principles of activity, namely, bilateral abduction and bilateral adduction; I have also pointed out that the control of these basal protective

band functions is always associated (by reason of a basal fixed animal neuro-muscular mechanism) with the muscular movement of the chest wall, which determines the passage of air through the air tube. Voluntary control of the vocal cords, then, always involves an associated chest wall action, making together a chest-vocal cord activity or function for the passage or stoppage of the movement of the breath through the larynx. And each chest-vocal cord activity, for abduction, or adduction, is capable of independent voluntary control. One basal chest-vocal cord activity, that for abduction, I have stated, determines the open position of the vocal cords for breathing, and the other chest-vocal cord activity, adduction, determines the closure of the vocal cords for the *prevention* of breathing. There are no other primary principles of action determining vocal cord movement.

But there exists the possibility of developing one complicated secondary principle of action of the chest-vocal cord mechanism, namely, that for the production of sound. These secondary movements of the chest-vocal cord mechanism are of course merely modifications of the primary animal chest-vocal cord movements, and are dependent on a specific voluntary control of those primary animal movements.

Two types of speech sounds are required for the production of speech. Each is produced by its own definite modification of the primary animal chest-vocal cord actions of abduction and adduction. Each requires a simultaneous combined action of the vocal cords for both abduction and adduction. This necessity arises from the fact that the vocal cords are, for the whispered as well as for the loud type of sound, held a certain distance apart, thus requiring a joint balance action of partial abduction and adduction. Whether the placement of the vocal cords for the production of each type of speech sound production, the "whispered" and the "loud" types, is determined by fixed cerebral-neuro-muscular mechanisms of action for each type of speech sound, or whether these vocal cord positions for speech sound production are the result of a secondary voluntary control over the abduction and adduction activities of the vocal cords, is as yet an unsettled question. However, the production of the required detailed placements of the vocal cords, of the oral articulative positions, and of the palatal placement, for each individual speech sound are undoubtedly secondary acquired voluntary activities.

The production of a speech sound requires the placement of the vocal cords in position for that sound, the cooperative creation of a mouth articulative channel, as well as the placement of the soft palate and, finally, the air from the lungs must, by chest compression, or expansion, be forced against and *between* the vocal cords, in order to create their vibration.

Thus the air in the larynx is caused to vibrate, and is (by chest compression) forced onward either out the mouth through the articulative channel, or, for nasal sounds, out the nose.

Since the child's acquisition of the capability of speech sound production depends on the imitation of words spoken in his environment, the resulting psychophysiologic habit of speech production is based directly on the effort to produce words and to unite them into sentences. A habit is also formed of producing individual speech sounds, but always as components of words. Since eventually the complete habit of word and sentence production always includes the ability to produce each speech sound independently, possibly this ability may be a natural necessity for acquiring accurately the production of words and sentences. However, possibly also the acquiring of a knowledge of the production of each individual sound is partly dependent on the fact that the speech machine can produce only one speech sound at a time. At any rate as the imitation of words and short sentences spoken in the environment continues, the child eventually learns to produce each speech sound independently of word production, and to place each sound where it belongs in new words.

VI. Demonstrating That the Prevention of Sound Production in Each Basal Act of Stammering Must Be Due to Vocal Cord Adduction

The production of a particular speech sound by the speech sound mechanism requires the placement of the articulative, the palatal and the vocal cord mechanisms in a position to produce the particular sound, and finally, the passage of air under chest compression against and *between* the vocal cords, causing them to vibrate; thus the air in the larynx is set into vibration. Then, the chest continuing its compression, the vibrating air in the larynx is forced, if it be an oral sound, out through the articulative mouth channel for that sound already produced; or, if it be a nasal sound, out the nose, behind the lowered soft palate.

It is thus to be noted that for the production of any speech sound the breathed air *must* pass between the vocal cords, and that for the stoppage of the production of a speech sound, when the producing mechanism is in action and the movement stopped as in stammering, the breathed air must in some way be prevented from passing between the vocal cords. In stammering, we must remember, the entire speech sound mechanism—chest, vocal cords, articulative organs and soft palate—is in action, with movement stopped and no air passing the vocal cords, and no sound forthcoming. This signifies that the passage of air between the vocal cords has been in some manner prevented. Nature's primary way of preventing such

air passage between the vocal cords (in animal functions) is by vocal cord adduction, and in so far as the vocal cords themselves are concerned vocal cord adduction constitutes the only way the passage of air can be prevented.

Nevertheless, it is now necessary to point out that vocal cord adduction does not constitute the only action of the speech sound mechanism capable of preventing the passage of air between the vocal cords even with each of its muscular systems in action. The existence of a second manner of preventing sound production, other than that of vocal cord adduction, has served to create confusion, which I shall now hope to clarify.

Except for vocal cord adduction, the only way in which air can be prevented from passing between the vocal cords when the speech sound mechanism is in action is by complete arrest of air movement through both the mouth and nose. In the production of a speech sound this double stoppage necessarily occurs in the production of the [p], [b], [t], [d], [k] and [g] sounds. In the production of each of these sounds the soft palate is raised to prevent the passage of air from the lungs through the nose. In addition, for the production of the [p] and [b] sounds the mouth exits are stopped by the closure of the lips; for the [t] and [d] sounds the mouth exits are stopped by the production of an anterior barricade by placement of the front of the tongue against the hard palate and the sides of the mouth; and for the [k] and [g] sounds the same effect results from the action of a similar posterior barricade.

In the production of speech sounds by the speech sound mechanism only these six sounds are produced, as has been said, by complete stoppage of air movement at the mouth. For the remaining approximately forty sounds the air is free to pass out of the mouth or nose. In conceiving of speaking these forty sounds, therefore, the only possible way for preventing the passage of air between the vocal cords is by the production of vocal cord adduction, and, therefore, vocal cord adduction constitutes the only possible means by which stammering on these sounds can be produced.

As to stammering on the [p], [b], [t], [d], [k] and [g] sounds, the stoppage of air at the mouth and nose can in no way prevent the production of vocal cord adduction as the actual cause of sound prevention in stammering on these sounds. Moreover, the detailed manner of stammering in attempting to produce these six sounds is not different from the details of stammering on any of the other forty sounds, as would be expected if the cause of stammering on them were different. Therefore, it seems clearly evident that the reason for sound prevention in stammering on these six sounds is the same as for the other forty sounds, namely, vocal cord

adduction. Therefore, we must conclude that every fundamental act of stammering must be determined by vocal cord adduction.

The above-stated facts, reasoning and conclusion, then, constitute the final word, in my judgment, as to the cause of the prevention of sound production in the individual act of stammering: every basal act of stammering consists of the psychophysiologic act of vocal cord adduction. This conclusion must now stand finally as a demonstrated fact, so far as I am able to determine. I believe that there is no way to refute its truth.

VII. The Psychophysiology of Stammering

Speech is a psychophysiologic function through which a physiologic mechanism of the body (that for breathing) is caused to produce individual physical speech sounds, and in such a manner as to represent human thinking. Each complete activity of the speech sound mechanism while talking results in a single speech sound. Combinations of such sounds constitute words.

Any competent observer of stammerers may learn to comprehend the following facts concerning this disorder in the production of speech sounds:

(1) A single act of stammering is a partial or complete psychophysiologic failure in the normal production of a single (or occasionally of two adjacent) elemental speech sounds during speech.

(2) In each individual act of stammering the musculature of chest, vocal cords, articulative organs and soft palate are in directed action as if to produce the individual speech sound intended to be produced but without the normal production of such sound. Evidently a psychologic misdirection of the speech sound mechanism prevents the normal production of the sound in mind. Although all muscles of the mechanism are in action all movement of chest, larynx, articulative organs and soft palate cease, and a normal sound is not forthcoming. Speech sound production is thus definitely and seriously thwarted.

(3) In the production of an individual act of stammering, in case the stammerer fails to make an attempt at normal sound production intended to offset his act of stammering, as he might do and often does, the vocal cords enter into complete adduction for an appreciable interval of time and no sound is produced.

On the other hand, if through the action of psychophysiologic conflict, an attempt to offset the act of stammering by an attempt at normal sound production occurs, vocal cord adduction is momentarily released, air passes the vocal cords, and a meaningless sound momentarily results. This constitutes a common manner of stammering.

(4) In learning to talk the child combines his effort at vocal cord movement to produce a word with a cooperating effort to produce the necessary articulative channel for each individual sound. One who acquires the voluntary act of vocal cord adduction of stammering produces the corresponding articulative position for the sound stammered on.

In the production of the primary act of stammering on any speech sound vocal cord adduction, by preventing the passage of air at the vocal cords, renders chest movement impossible. Nevertheless, the stammerer tends to struggle on its production, which struggle is usually carried along to the articulative muscles producing the corresponding volun-

tarily produced mouth position for the sound being attempted, which continued struggle constitutes a secondary act of stammering.

The primary act of stammering concerns vocal cord adduction, the secondary acts of stammering concern the organs of articulation.

(5) The muscular struggle in the acts of secondary stammering always includes an upward pulling by the extrinsic muscles of the larynx on the thyroid cartilage, and, therefore, all mature stammerers have gradually developed a more or less vigorous habit of upward holding of the thyroid cartilage and, therefore, of abnormal stretching of the vocal cords. This abnormal habit action, rendering the pitch of the voice unduly high,, is carried out in normal as well as stammering activities of the vocal mechanism. This habit of up-pulling of the thyroid cartilage destroys the normal method of superinducing low vocal pitch. One may feel the upward movement of the tip of the thyroid cartilage in every mature stammerer.[1]

VIII. SUMMARY

A. *The Basal Psychophysiologic Facts of Laryngeal Action*

The protective bands of the larynx (the vocal cords) can be subjectively appreciated neither as to their existence nor their movements.

The basal primary movements of the protective bands of the larynx (vocal cords) consist of (a) bilateral abduction, and (b) bilateral adduction.

Their movements are dependent on fixed cerebro-protective band neuromuscular mechanisms, primarily for animal purposes.

When the larynx is acting independently of contact with other organs (base of the tongue, etc.) each of these primary vocal cord activities, bilateral abduction and adduction, is, by reason also of fixed neuromuscular mechanisms, *always associated with either chest wall compression or expansion for control of air in the lungs.*

Each of such basal activities of the protective bands, abduction or adduction, is controllable voluntarily as well as involuntarily.

The animal purpose of protective band abduction is the opening of the protective bands for breathing; and the animal purpose of protective band adduction is their closure for the prevention of breathing.

The production of the animal activity of adduction, as well as of abduction, is especially concerned with involuntary or voluntary production of cough and compression of the abdomen, and the solely involuntary act of sneezing.

One gains voluntary control of the movements of the vocal cords only through primarily setting the chest musculature into the action of compression or of expansion, as if for breathing.

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[1] See Kenyon, Elmer L. Relation of oral articulative movements of speech and of extrinsic laryngeal musculature in general to function of vocal cords, *Arch. Otolaryngology*, 1927, 5, pages 15 to 20 of reprint.

The subjective voluntary thought-purpose of producing chest compression or chest expansion coupled with the intention of breathing, results in voluntary vocal cord abduction. Also the voluntary subjective purpose of producing chest compression or chest expansion, coupled with the intention of *preventing breathing*, results in vocal cord adduction.

B. *The Psychophysiology of the Production of the Sounds of Speech*

The production of each speech sound is developed from the basal animal chest-vocal cord activities of bilateral abduction and adduction.

The production of a speech sound, either "whispered" or "loud," demands the simultaneous, combined, partially balanced action of both abduction and adduction of the vocal cords.

The proper position of the vocal cords for the production of any speech sound is secured through the subjective purpose of producing that sound.

Each complete action of the speech sound mechanism results in the production of a single elemental speech sound.

Vibration of the vocal cords for a speech sound is secured by the impact of the breathed air against and *between* the vocal cords, already in position for a particular sound.

The air thus caused to vibrate, if the sound be oral, passes out the mouth through a specific channel created for that sound, or if the sound be nasal, out through the nose.

The development of speech demands the acquisition through imitation of the habitual ability to produce the sounds of many words and many short sentences, every elementary speech sound of which words are composed, and, finally, to build any combination of sounds or syllables.

C. *The Psychophysiology of Stammering*

Stammering is concerned only with the action of the larynx when not in operative contact with other organs (base of tongue, etc.).

Stammering is fundamentally a specific disorder in the psychophysiologic accomplishment of the vocal function of speech. The basal act of stammering consists of the following: While the mechanism of speech sound production is in action for talking, a compulsory interpolation (for an act of speech sound production) of a normal but misplaced psychophysiological act, namely, vocal cord adduction, is caused to occur. This prevents the passage of air at the vocal cords, and, therefore, prevents sound production, thus distorting the act of talking. This misplaced psychophysiologic act, voluntarily accomplished, possesses through repetition, a strong element of habit, and constitutes the foundation for every basal abnormal

act of the disorder. From my standpoint, this is a statement not of theory but of observed fact.

In each basal act of stammering the musculature of the speech mechanism *is still in action*, although *movement* of the mechanism has ceased; air cannot pass between the closed vocal cords and a speech sound cannot be produced.

Vocal cord adduction, which constitutes the psychophysiologic basis of stammering, is possible of voluntary production by every normally intelligent human being, child or adult.

Actual proof is developed that the prevention of sound production with the muscles of the speech mechanism in action, as in stammering, can be accomplished only through complete vocal cord adduction.

The primary muscular abnormal action of stammering consists of action of the chest musculature for an impossible movement of the vocal cords, and the secondary abnormal action consists of an articulative struggle superinduced by the failure of action in the primary struggle. Eventually both acts, primary and secondary, become strong habits, and include permanent upward pulling of the thyroid cartilage (in both effective speech and stammering).

Recall the violent struggles of the severe stammerer, and yet with air passing the vocal cords not at all or only in spurts (due to his abortive attempt at normal speech production) and with this picture in mind, realize that the prevention of the passage of air between the vocal cords in any act of stammering is possible only because of the cerebro-chest-vocal cord act of adduction. Then you possess the essential picture of the psychophysiologic nature of each stammering act, namely, the substitution of vocal cord adduction for the production of an individual speech sound. (See Section 6 of the present Essay.)

MEMO

The News and Announcements section provides a "bulletin board" for the profession, and items concerning new programs you are developing, new positions that have been created, brief summary statements of surveys you have completed, important developments in related professional associations, changes in positions held by our members, etc., should be sent to Dr. Ernest Henrikson, Department of Speech, University of Denver. *Deadline for the March issue is January 1.*

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MODERN APPROACH IN THE STUDY OF APHASIA

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I.

A few years ago I became interested in making psychological studies of patients with unilateral frontal lobectomies.[1] In studying the literature on the functions of the cerebral cortex, it soon became evident that disagreements as to theories and facts were most intense with regard to aphasia. The approach in my study of the behavior changes resulting from frontal lobe lesions was not primarily focused on speech disorders, but certain experimental data obtained in my study could be applied directly to the interpretation of certain disorders of speech. I claim, however, no originality for the conclusions which were reached, because what I have to say is expressed in all modern trends of thinking on aphasia.

Before making a statement as to what aphasia is, from the standpoint of my observations, I wish to make a few remarks about the errors or fallacies which have been made in many of the observations and theories on speech disorders of the type under consideration. These criticisms are especially pertinent in view of the fact that, as Grinker (4) points out, there has been little progress in the field of aphasia since it was first described. This has especially characterized the work on speech disorders prior to this century. The fallacies which have impeded progress in the understanding of the nature of aphasia have been pointed out by many modern experimenters in this field.

The first fallacy in the study of aphasia has been the emphasis, especially before 1900, on strict cerebral localization of different forms of speech disorders. Head refers to this school of thought as the diagram-makers. It is true that lesions producing certain disturbances of speech are roughly localized in certain brain areas. There is, however, no such strict localization of speech disorders as has been postulated by such workers as Bastian (1). I am going to point out later that even lesions in the non-dominant hemisphere may produce symptoms analogous to disorders which have been placed in the category of speech disorders by some experimenters.

The second fallacy in the study of aphasia has been the names or classifications used for the disorders of speech. Classifying external symptoms is only the first step above complete ignorance of the subject. Such classifications of

[1] The patients were furnished by Dr. Loyal Davis from his private practice, and the research was conducted with him.

symptoms, if accepted as explanations of the phenomena, very often obscure the functions which produce the symptoms. Head (6), in making his classifications, warns against the acceptance of them as explanatory terms. Nevertheless, they have already been raised to the status of explanatory principles by some textbooks on neurology. Weisenburg and McBride (7) make the statement that there are 28 different classifications of speech disorders. This reminds one of the classifications of mental disorders introduced by Kraeplin. They look nice when entered on the patient's chart but yield no insight into the functions responsible for the neurosis or psychosis. The understanding of the disorders is more nearly approximated when the classifications are forgotten and the underlying causes for the disorders are studied.

The third fallacy in the study of aphasia has been the inadequate nature of the observations made on aphasic patients. Most of the studies prior to 1900 and some up to the present time have used inadequate experimental methods. This has led to the belief that many kinds of pure speech disorders exist. Goldstein (3), in 1910, pointed out that where refined experimental methods are used, many functions are found to be disturbed in cases which are clinically diagnosed as pure types of speech disorders. I have had a patient referred to me as suffering from motor and sensory aphasia and alexia. The patient's mental powers were said to be intact: she could play pinochle, keep score, etc. More thorough examination showed that all phases of her performance were disturbed. Contrary to the statements of the sister of this patient, she was unable to play even simple games, and her arithmetical proficiency was only of the simplest kind. I also have had patients who clinically showed no disturbance of functions, but none of these patients failed to show positive symptoms when refined experimental methods were used. One such case, a cost accountant for a large corporation, with a right frontal lobectomy, was able to go back to work three months after the operation, and has been doing his work adequately for the last two years. He showed a number of disturbances, however, when the proper experimental methods were used.

The fourth fallacy to be pointed out is represented by the fact that many observers fit the clinical or experimental data into their own methods of thinking, without attempting to make an exhaustive experimental analysis in the absence of pre-conceived notions. It is only too easy to interpret an experimental fact in terms of one's own theoretical bias. As an example, a patient is unable to pick out hues in the Holmgren wools test for color vision. The easiest explanation would be that the patient is color blind. However, other tests show that the patient is not color blind. Another explanation which has

been offered for such behavior is that the patient has an aphasia to certain words, especially those pertaining to color. More elaborate tests show, however, that the patient's behavior can be explained in still another way. Gelb and Goldstein (2) have been led to the conclusion that the behavior of such patients is due to a disturbance of abstract or categorical behavior. Thus the same experimental data are amenable to a number of different interpretations. As I shall point out later, some of my observations could have been interpreted in a number of different ways, and might have fitted into anyone's biased category.

II.

To emphasize the viewpoint which will be presented against the background of different modes of thought, I want to make a brief historical survey of explanations as to the nature of aphasia. The first explanation of aphasia and kindred disorders was made in terms of certain faculties which, according to Gall, were localized in definite areas of the brain. This form of localization was superseded by localization in terms of sensory and motor areas of the brain and their interconnections. This viewpoint also emphasized mental content, but for the faculties such terms as sensation, images, and perception were substituted. More recently there have been attempts to analyze speech disorders on the basis of functions. One of the approaches has involved the analysis of different forms of speech. Finally, and this is true of Jackson, Goldstein, Head and others, there have been attempts to ascribe the pathology of speech to certain functions which are disturbed. These functions or mechanisms may be entirely different from the symptoms shown in the speech disorder.

We now come to a very fundamental attitude in connection with the interpretation of speech disorders, and that is that the pathology of speech due to brain lesions is a symptom of the disturbance of certain functions or mechanisms. Although the speech disorder may be the most dramatic change produced in the patient by the lesion, it may give little insight into the kinds of function or mechanism which are disturbed. In order to know what these functions or mechanisms are it is necessary to analyze all (or as many as possible) of the disturbances shown by the patient. If certain similarities of function appear on tests of diverse nature, then it is possible that all of the forms of atypical behavior in these situations are due to the pathology of a certain underlying mechanism. If this viewpoint is accepted then the problem of aphasia becomes a problem of the study of certain mechanisms which produce a deficiency, or what might be called release phenomena, in the individual. For that reason the analysis of speech alone

is not sufficient, and possibly experiments which have nothing to do with speech may yield more information as to the functions or mechanisms which are disturbed.

The nature of the mechanisms which are involved following the destruction of cerebral tissue cannot be postulated with any degree of certainty. Before mentioning the results of my experiments in this connection I would like to draw attention to the similarity of a function postulated by Hughlings Jackson, Kurt Goldstein, and Henry Head. Jackson was among the first to postulate the different levels of functioning of the nervous system. This same concept appears in Goldstein's work when he speaks of abstract and concrete behavior. Head's notion of symbolic formulation closely resembles that of Goldstein's categorical or abstract behavior. Although there is general agreement that a more elaborate function is destroyed following the cortical lesions, the analysis is not complete; I believe that Jackson's highest level of function, Goldstein's abstract behavior, and Head's symbolic formulation consist of possibly a number of functions. I shall, therefore, describe some of my results with an attempt to show how they may be related to aphasia and to name the possible functions which are involved.

Threshold of Perception. Twelve cards (with meaningful and nonsense words, and geometrical figures) were exposed with a Dockeray tachistoscope. The patients with cerebral lesions took a longer time to perceive the stimuli. This is analogous to the reactions observed in aphasics after recovery. Head states that when an aphasic patient recovers the symptom which persists is the long reaction time.

Interpretation of Ambiguous Figures. Six cards with ambiguous figures are presented one at a time to the patient and he is asked to name as many meaningful objects as he can see in the figures. The patient with cerebral lesion does not see as many objects in the figures as the normal individual, although, as far as can be determined, the patient has no difficulty in naming actual objects. Nevertheless, this deficiency approximates that of visual agnosia.

Memory for Objects. Sixteen objects are presented visually for 15 seconds and the patient is asked to name them from memory. The patients with cerebral lesions are unable to name as many of the objects as the controls. This does not seem to be a function of so-called memory, but the inability to remember the objects by making certain logical categories. As an example, spoon, knife and fork can be remembered normally because they are assigned to the category of kitchen utensils. If this ability is absent the memory for objects will suffer. This inability fits into Head's postulate that in aphasia symbolic processes are affected and is relevant to Goldstein's notion of categorical behavior.

Jargon. None of the patients showed jargon in his speech in its purest form, but many patients, including those with a right cerebral lesion, showed increased talkativeness which, although dealing indirectly with the context of the topic under discussion, had some features of redundancy. Such mild nonsensical talk is often found in its extreme forms in aphasia, and could be placed in this category. Even in the mild form Head has called such a disorder semantic aphasia. This form of behavior can be explained in other ways. It may be taken to indicate a release phenomenon from the higher cortical centers, or a method used by the patient to cover up his apparent lack of understanding.

III.

From these results and results of other tests it was concluded that at least the following functions were disturbed in cases with unilateral lobectomies: 1. The threshold of perception is affected so that it takes a longer time for an individual to apprehend a situation. 2. The number of features which can be perceived in a situation is below normal. 3. The ability to shift from one stimulus to another is decreased. 4. The ability to perceive logical relationships between objects deteriorates.

These functions which are disturbed in cases of cerebral lesions must affect all forms of behavior, whether verbal or not. Speech disorders, from the standpoint presented here, would follow because certain underlying functions on which all behavior is dependent are affected. To understand the manner in which the functions are related to speech disorders requires a knowledge of all the functions which are destroyed, and how these mechanisms are responsible for the appearance of normal speech. In either field the amount of information is not sufficient to yield even adequate approximations to answers to these questions. For the problem of re-education in the field of speech pathology one conclusion is clearly indicated, that possibly the re-education of aphasics will eventually require an emphasis on the mechanisms which underlie the production of speech, and not, or to a lesser extent, on training in speech itself.

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NEWS AND ANNOUNCEMENTS

(Continued from Page 336)

Treasurer) and Dr. Raymond Carhart, who is now Program Chairman by virtue of his recent election to the office of Second Vice-President. Council action at Chicago directed that all correspondence concerning inter-association convention activities be carried on through the ASCA Secretary-Treasurer, Dr. Morris.

The Inter-Organization Convention Committee serves as a clearing center for communications and plans involved in cooperative conventions, such as those held in Detroit in 1941 and in Chicago last year, in which the ASCA met with the NATS and its affiliated groups. In 1940, it will be recalled, the ASCA met at State College, Pennsylvania, with the American Psychological Association. For many years the ASCA has followed the policy of holding its conventions in conjunction with the annual meetings of closely related, and usually larger, professional organizations. Speech correctionists have much in common with workers in general speech, psychology, education, child welfare, medicine and allied fields, and cooperative conventions have proved to be advantageous to all concerned. The ASCA was originally organized at a meeting of the NATS, and ties between these two groups have always been particularly strong.

That organizations with interests kindred to those of the ASCA also are moving ahead with dynamic, forward-looking programs is evidenced in the special attention given National Hearing Week (October 24-30) by the American Society for the Hard of Hearing. Extensive publicity was given the week through radio programs, newspaper and magazine articles, window displays, and the like. Further, the American Society for the Hard of Hearing is already cooperating with the Army, the Veterans' Administration and the United States Rehabilitation Service in preparing to help soldiers and sailors returning from the battle fronts with impaired hearing. This is being promoted through classes in lip reading, planned recreation, information services and practical psychology.

The *Journal* is happy to announce an activity in another field of kindred interest to speech correction, that of general semantics. *ETC: A Review of General Semantics* appeared for the first time in August. This is the official organ of the Society for General Semantics and will be issued as a quarterly publication under the editorship of Dr. S. I. Hayakawa, Department of Language and Literature, Illinois Institute of Technology, Chicago.

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WORKING WITH SPEECH DEFECTIVES IN PUBLIC SCHOOLS*

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I.

Before one can help a handicapped child it is necessary to get at the causes of the difficulty, to find out what things have prevented the child from growing and developing normally, and to do what can be done to modify conditions not favorable to growth. In order to do this the speech correctionist in a public school must investigate the resources that are available for helping the child, and then offer as much guidance and direct aid in the way of speech therapy as she can.

To the speech correctionist the school nurse is an invaluable person. She can usually obtain a complete medical history, investigate the home situation, and arrange necessary medical care through social agencies when parents are not able to provide for it. She can also work directly with the child, or with his parents, to help the child develop hygienic habits which give him a better chance for adjustment.

The importance of the school nurse in relation to public school speech correction can be illustrated with the case of Jimmy. Jimmy's mother did fine laundry and housework by the day to make a home for Jimmy. She had to stop school in the third grade but she wanted Jimmy to have an education. Jimmy liked to draw and he loved to be with the other children, but he couldn't learn to read or to spell or to speak very well, because he couldn't hear adequately. The nurse arranged an otological examination for him and, upon the advice of the otologist, she arranged for his tonsils and adenoids to be removed at the Children's Dispensary, so that when the speech retraining program was started we knew that Jimmy's hearing was at least as good as it could be.

The school nurse helped with Bob, too, who was a severe stutterer, fourteen years old. He walked the mile between home and school four times a day, and worked in his father's store or passed out bills after school. He began to make better progress after the nurse arranged for him to have his lunch

* As a practical response to the rather widely expressed desire of *Journal* readers for occasional articles that present the firsthand experiences and workable procedures of speech correctionists, especially those working in public schools, the Editor asked Miss Schuell to write this paper. Those on the Editorial Staff who read it voted for its publication. Readers who feel that the *Journal* should publish more material of this type are urged to attempt in their own way to write articles like this. The Editorial Staff will give careful consideration to them, and will accept as many as can be published without distorting the over-all balance of the *Journal* contents.—The Editor.

at school and to have a rest period in the middle of the day.

In the writer's school system there is also a psychologist, and she has proved to be of service to the speech correction program. Her report and the recommendations she makes to teacher, principal and parent help them to understand the child's difficulties and to set about helping him more intelligently. Sometimes she recommends a modification of the child's school program, so that there is less pressure upon him, so that he works at a level where he can succeed, and the burden of continual failure is lifted from him. Sometimes she shows parents how to make adjustments which help both them and the child.

To cite a specific case, a young foreign mother tried to learn English, but gave up because her husband learned it so much more quickly than she. The psychologist pointed out to her that her husband's wider contacts, and his necessity for using English daily in his business, had enabled him to learn faster, and that his more rapid progress did not indicate a lack of intelligence on her part. Thus encouraged, the mother agreed to take some lessons and try again, this time with the specific objective of helping her five-year-old son adjust better to the English-speaking world in which he felt himself an alien.

II.

It is not only among the members of the school staff, however, that the speech correctionist can find persons whose cooperation will make her efforts more effective. Outside the school there are, first of all, the parents of the children with whom she works. It is important for the correctionist to know them as well as she can. Almost always they want to help the child if they know how.

Helene was six years old. In the first grade she would not talk or play at all. She did what she was told to do, and she read aloud when called upon to do so, but in a voice that was almost inaudible, and the words, characterized by omissions and substitutions, were almost unintelligible. When brought to the clinician she sat and cried silently. She was in good health, there was no hearing loss, no structural abnormality, and except for some language retardation, she had normal intelligence. The mother was interviewed, and it was found that the child was the "in-between" one in a large family. They were poor, but thrifty and hard-working. Effort was concentrated upon keeping the older children in high school, helping the oldest son, who was working his way through college, and the necessary care of the small baby. Helene just didn't come in anywhere. She was big enough to take care of herself, but not big enough to be a part of the plans and activities of the

others. She even took being left out for granted; she had no other expectations, at school or anywhere else, and knew no other kind of behavior. The family did not realize this, of course; they just thought that she was shy. The new program set up for her included having the older sister read to her, a game of checkers with her father before bedtime, and being allowed to help her mother with small tasks that she could do, being praised when she had done them as well as she could. The mother also managed to buy her a new snowsuit, some pretty school dresses and hair ribbons. She stopped crying about coming to school, and gained enough confidence that she was participating freely in the schoolroom and on the playground long before more than one or two sounds had been corrected; and the omissions, for the most part, cleared up of themselves.

David was a fourteen-year-old stutterer, with an I.Q. of one hundred and thirty-eight, and his home presented a different kind of picture. It was a beautiful house, comfortable and livable. David had a room of his own, a place for his books and hobbies, and a dog. His father was a professional man, and his mother was an attractive, intelligent, and poised person, and David was rather proud of them both. One of the first things that the clinician learned from them, however, was that because it had been observed that David did not stutter when he was angry, he was urged to pretend that he was angry and try to force the word out. Faked blocks by the clinician, showing the tensions resulting from forcing, some of which were already characteristic of David's speech, were enough to make them agree to abandon this method. It was discovered further that both parents held exceedingly high standards for David, that they wanted him to be perfect, really, and were worried lest he should not be. This predisposed them to worry about the stuttering, which predisposed David to worry about it, too, to try to avoid it, to hide it. Since he could do neither very well, he avoided reciting, and making contacts with his schoolmates in a new school. The parents were intelligent enough that they saw the harmfulness of this, and set themselves to try to adopt a more reasonable and realistic attitude toward the boy. They were urged to adopt an attitude of confidence rather than anxiety toward him, at least, and if they did not feel that it was successful, to send him to a nearby university speech clinic the following summer. The third aspect of the situation which was thought to need changing was their exaggerated idea of protecting him from undesirable associates, so that he was allowed little freedom. It was agreed that he should regularly invite classmates to the house, and be allowed more liberty in doing things with them as well. David's subsequent happier adjustment was

commented upon by his counsellor and other teachers, and the following fall he entered a general public speaking class in high school, and got along all right.

Parents are not always able to do what is best for the child, unfortunately, even when they want to. The father of an eleven-year-old spastic daughter was willing to do anything in the world for the child except to control his own emotional attitudes toward her. He never quite got over the idea that anything she didn't want to do was bad for her. On the few occasions when, under strong pressure from the clinician and the head of the University Clinic, he followed through a planned course of action, he ran the entire gamut with the child of cajolery, bribery, pleading, outburst of temper, threats, assertion of authority, then more bribes and entreaties and remonstrances which ran, "You don't love your papa; do you want to break your papa's heart?" And so on. Special arrangements were made to send her to camp with some of her friends; the director, skilled in physiotherapy, agreed to give her special help. She was sent home after one day. She attended the University Clinic for two summers, and was separated from her parents for eight weeks. After she had tried there, unsuccessfully, all of her most cunning devices for getting attention and having her own way, there was a good deal of progress in developing more adequate attitudes, but the regression when she returned home was fairly complete, and when last seen there did not seem to be too much hope for her.

III.

In any school there will be found a good many sympathetic and understanding classroom teachers whose wise and generous cooperation is one of the greatest aids to the correctionist. Helene, the "in-between" girl mentioned above, was fortunate in having a teacher of this kind, who, although she had forty first-graders in her room, always took time to give the little girl the quiet and friendly recognition she was so hungry for, to draw her gently into activities, and who found it an occasion for rejoicing when Helene began to respond, to volunteer shyly, and finally to participate without fear. Such a teacher will help the child to carry over and use each new gain which comes from the speech correction program, and her importance cannot be overestimated.

A school is a children's community, an important part of the child's world. Too often the child with a speech defect withdraws from it, and from fear of failure, fear of ridicule, fear of rejection, holds himself apart from all of it. Consequently, it sets a great deal right for the child if the clinician or some teacher can draw the child into some activity, so that

he has something to do of his own, feels necessary, and gets the feeling of belonging there.

John, a severe stutterer, was taught to run the movie machine. This activity was chosen at the beginning, first, because he had some mechanical ability, and liked doing it; secondly, it was a privilege all the boys enjoyed having, and so carried some prestige; and thirdly, John felt safe. He didn't have to talk, and his fear of speech was very great. One day, however, the thing broke down during a movie. Everyone who knew John was amazed when he calmly called for lights, grinned, and explained what was wrong, fixed it, called for lights out, and went on. Later, in high school, John had a long and honorable dramatic career.

David, intelligent and sensitive, and in a new school, had a great fear of rebuff, and did not know how to make friendly overtures to his classmates. He was never entirely at ease with them. He was given charge of lights for a school production. He was called in to a preliminary technical rehearsal, helped to set up equipment and run trials, and made the final charts himself. The next day the entire crew was called, and the only directions they were given were that David had the charts; they should ask him. All through dress rehearsal questions rang through the auditorium: "Hey, Dave, what goes here? Where's the blue gelatin at? Can I run this spot, Dave? Have you got the hammer?" and so on. Dave yelled back with calm efficiency, and when he said, "Some one get a ladder," three boys ran. At the end of the fifth performance, Dave was one of the gang, and he hadn't had time to bother about whether he stuttered or not.

Lawrence had a cleft palate, was under-nourished, underprivileged, and had no doubt experienced real rejection. When he came to a new school in the eighth grade he did a good job on his speech, working privately with the clinician, but he could not be induced to recite in class or have anything to do with his classmates. All he hoped for, really, was to be as unobtrusive as possible, and to escape notice that he was sure would be unfriendly. A voice and articulation test was given to the entire class. For the articulation part the loud-speaker was set up, and they were allowed to talk through a microphone set in the hall. They were all interested, and the discussion which followed was made just technical enough to arouse a little scientific curiosity. Their test sheets were analyzed, various articulatory defects explained, and Lawrence's special problem and the work that he was doing on it was discussed impersonally along with the rest. He sat stunned, but the next week consented to arrange with the social studies teacher to read a paragraph he had prepared before the class. One boy in the class came to the correctionist afterward and

said, "I just want to tell you what a swell job I think Lawrence is doing with his speech. He's been in my class ever since we were in the third grade, and this is the first time we've ever been able to understand him." The other children, too, thought that he was doing a swell job. The next semester Lawrence was actually elected president of his sponsor group.

The small child, too, needs to be made to feel at home in the school. Sahag, a little Armenian boy, understood English when he came to kindergarten, but the school environment was so foreign and strange to him that he was afraid to try to talk. His first response was a whisper, which he continued to use for about three weeks. During this period he made all the unvoiced sounds, but refused to try a single voiced one. At length he began to use voice with the clinician. On the same floor as the kindergarten were the music room, the art room, the shop, and the cafeteria. We visited all of them in turn and came back and talked about them. In the art room there was a jaunty paper cast of a giraffe, made from a clay model and painted. This pleased Sahag because they had had a story about a giraffe in the kindergarten and he had seen pictures of them. The art teacher allowed him to take it back to the kindergarten to show the other children. A few days later the art teacher strolled into the speech room where Sahag was drawing on the board. He said, "Hello, Sahag, what are you drawing?" Sahag didn't turn around, but he answered aloud and easily, "A train." Shortly after this he said his number aloud when they were counting in the kindergarten, named colors aloud when his turn came, and answered his teacher's friendly "Good-by" with an audible "Good-by."

IV.

Among the resources available to the speech correction teacher there are other things besides nurses, psychologists, teachers, parents and school activities. There are also the paraphernalia and techniques—the "bag of tricks"—that can be used to liven up her work and make it more effective. Children like to come to the speech room, for example, if it is a friendly and an interesting place. Almost anything at all which would interest a child can be used to make him forget his fears and anxieties at first, and later they can provide motivation for a speech attempt. A shelf of illustrated books, a drawer or shelf with picture cards, a table with a collection of random objects, never too many at a time, all are useful. In the writer's room a bowl with little live turtles which could be fed, contributed by some of the older boys, was an object of fascination for a year. Valentines in season, props left over from a children's play, colored chalk and the wonder-

ful privilege of drawing on the board, all furnish stimulation or a starting point for a speech lesson.

A turtle can be useful to a child working on *r* or *l* or *t*. The table can hold objects which have an *r* or an *s* or any other desired sound in the name, or a mixture of those which have and those which do not have the desired sound, to be discriminated.

Helene's first *r* came in naming a gaily-painted *butterfly* left over from a play; another came soon after when she wanted colored chalk to *draw*. She had not been able to make the sound in isolation at all.

The child can show you pictures he *likes*, or tell you to *look* at them; he can name them with *there is*, or *that is*, for the *th* or the *z* sound, or simply name them with *the*; he can *choose* the ones he likes best, he can start with *see*. He can win the card if the sound is a good one. Infinite variety is possible, and much monotony is avoided during the period of establishing the sound.

Tommy was a six-year-old who had a curious and inquiring mind, but he had been sick a lot, and his mother was deaf, so that he had not had much stimulation for speech. He knew what things were but he did not know the names of them. Sometimes he knew the name but was not sure enough of how to say it to try. He talked very little in the fall, and the first responses came hesitatingly and uncertainly. When he had overcome his first fear of making mistakes he began to ask, "What dat is, what dat is?" over and over. He seldom forgot a name he had asked for. In the next period almost anything could set him off, and he would prattle to himself contentedly, usually demanding attention only when he wanted to know a name. The correctionist allowed him to come in during the noon hour, and after he had had his lesson she started him off with something to look at, then pretended to be busy while Tommy made up for some of the time he'd lost not talking. This is a record of Tommy sorting through valentines:

Tommy: There's all the animals. An Indian, a Valentine Indian. Here's a lion. He's crying. He's a sissy. You sissy. O-o-o-oh, my! Here's a little valentine. Here's one. Here's a little boat. Here's a big boat. I'll stand this one up here. What this is she has? Her's makin' a flower. Let's see dis one. Who's dis?

Clinician: Cupid.

Tommy: Cupid? A little baby? How he walk? He flying. He have wings. I'll stand this one up here. What this is she has? Her's makin' a girl—open it back at a valentine. See it. See it. See it. See it fall down. See it inside. See it.

Clinician: A cowboy.

Tommy: A cowboy. A little pussy, and he hanging up. See it. See it. A funny one. A teacher. A funny teacher. See it.

Clinician: A teddy bear.

Tommy: A teddy bear. See it, it's crying. A little valentine. See it. Don't look at it this way, look at it that way. See little chicken. See, dey makin' valentine balls. See it inside, O-o-o-oh, nice. Here, I like dis better. And I like dat little kitty. Where did he go? I don't know why this tent has hearts on it. Where his bow and arrow? What this girl doing?

Clinician: Dancing.

Tommy: Dancing? (Makes her dance, and sings, using vowel sounds.) O-i-e-i-e-i-o. I don't have this one, I don't have it, dis ain't it. (Bell rings.) I think I have to go, huh?

Clinician: When the children come in.

Tommy: When the children come in? I gonna see. I got the same one that you do. See, here one. What this one do? (Children enter.) Can't play. Goodby. (Skips out of room.)

Tommy is seven now, and there is no sign of any retardation. He is a good reader. The psychologist dismissed him last spring, as needing no further special help, except in speech; the *r* and *l* sounds are not entirely established yet. At six the psychologist had given him the benefit of a doubt when she allowed him to enter school at all; he seemed so retarded that it was not at all certain that he could get anything from the experience.

Another simple and useful device is for the correctionist to write the child's name on a piece of paper at the beginning of the lesson, and then jot down the sounds or words they are working on. Red-pencilled stars after successes furnish motivation and give the child a record of success which he is proud to show to teacher and parent. Most of the children have had few enough successes to show, and they value these papers. They will often remind you about their papers, if you seem not to be remembering. Of course, this is also a time-saving device for showing the teacher and the parent just what the child is working on, and what can be expected of him. Notes can be added about assignments he is to do outside, and this sometimes helps the child to remember, too.

V.

A child grows. A worried, anxious youngster becomes confident, happy, and finds his place with the rest. You never know just how or why, although you can see when it begins to happen usually, and then one day the signs are quite clear. These things have seemed to help some child at some time, which is reason enough, perhaps, for writing them down.

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THE EFFECT OF HEAD INJURY ON HEARING[1]

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I.

A study of the function of each cranial nerve is of vital importance in relation to an injury to the head. Cranial nerves are damaged by such an injury in inverse proportion to their length. The shorter the nerve the more apt it is to be damaged. This is quite independent of whether the skull is actually fractured or not, and is due to the rocking movements of the brain within the cranial box and the tension which these movements impose upon a short nerve. The VIIIth nerve, pursuing the shortest distance of any of the cranial nerves between its point of exit from the brain stem and its entrance into the internal auditory meatus, is the one most frequently damaged by the rocking movements of the brain as it floats in the cerebrospinal fluid. Thus the hearing function can be damaged by stretching, tearing or torsion of the VIIIth nerve.

The hearing can also be damaged by hemorrhages into the perilymphatic space in the basal coil of the cochlea near the round window. It has been experimentally demonstrated that these hemorrhages are the direct concussional effects of the blow and occur whether the skull is actually fractured or not. Of course, if there occurs a longitudinal fracture of the temporal bone, running along its anterior border, these hemorrhages into the basal coil of the cochlea are especially prone to occur. They can, however, occur without any actual fracture. The hearing defect caused by these hemorrhages is of the perceptive type of deafness and involves the upper tonal range because the higher tones are localized in this area.

In addition to the effects of head trauma on the hearing function due to the tearing or stretching of the VIIIth nerve or to hemorrhages into the basal coil of the cochlea, the hearing can be partially damaged in still another way. If the above mentioned longitudinal fracture of the temporal bone actually crosses the tympanic cavity, dislocates the ossicles and ruptures the drum membrane, it frequently causes an interference with the free movement of the ossicular chain and middle ear adhesions resulting in a conductive form of deafness. This, superimposed upon the effects of the basal coil hemorrhages, results in a combined type of deafness of varying degree.

The above-mentioned pathology produces only partial loss of hearing. Total deafness of one ear may ensue from a so-called transverse fracture of the petrous pyramid. This fracture, fortunately relatively rare, begins in the posterior cranial

[1] Read before the American Speech Correction Association, Chicago, December 30, 1942.

fossa, crosses the pyramid at right angles, and, in so doing, destroys the neuromembranous elements of the cochlea and vestibule resulting in complete loss of cochlear-vestibular function.

From the foregoing it will be seen that the pathology which results in defective hearing is restricted to the VIIIth nerve itself or the cochlear contents. Because of its wide distribution and its bilateral representation, it is inconceivable that the hearing function can be materially damaged by direct injury to the brain substance of the center for hearing. To all intents and purposes, therefore, the pathology which causes hearing defects is peripheral in location.

II.

The functional examination for hearing is not reliable in the early days after injury, because of shock and the disturbed condition of the sensorium. The quantitative functional examination must be delayed until the sensorium has cleared and, even then, the results in individual cases are hard to evaluate. The examination of the hearing function is entirely subjective in character and we are entirely dependent upon the truthfulness and cooperation of the patient. We often have no knowledge of the individual's hearing function prior to his injury, and this history is particularly important in older individuals. These cases are almost always medico-legal and one must be continually on guard for malingering. Reliable deductions can be made only after studying a large number of cases and after making repeated examinations in each individual case. The hearing tests which are made soon after accident are qualitative only and are made solely for the purpose of excluding a fracture of the otic capsule.

The functional hearing examination should include a careful past history for such diseases as scarlet fever, measles, mumps, influenza, typhoid, syphilis, and previous attacks of otitis media. It should include a work history to evaluate the influence of a noisy environment, especially in middle age and beyond. It should include an investigation of the drug habits of the individual with particular reference to quinine, the salicylates, and lead exposure and an inquiry about familial deafness. It should include careful and oft-repeated tests with the voice, and tuning forks and the audiometer. It is not sufficient to use only one of these methods. The use of both tuning forks and the audiometer, as well as the voice, will determine whether there is a proper correlation between these various tests. It should also include a careful examination of the vestibular mechanism. Because the pathology which damages the hearing function is peripheral and because there is a close peripheral association of the cochlear and vestibular apparati, it is rare indeed that we find a traumatic loss of hearing not

associated with some signs and symptoms of vestibular disturbance. I do not say that it can never occur, but if I do find a hearing loss not associated with a vestibular disturbance I am inclined to be very suspicious of its traumatic origin.

III.

In order to study the hearing function in various types of skull fractures, I recently surveyed 211 cases of proved skull fracture. I studied these cases in the following groups:

(1) Fractures involving various regions of the vault, face, or sinuses, but not involving the base—49 cases.

(2) Unilateral longitudinal fractures of the temporal bone—112 cases.

(3) Bilateral longitudinal fractures—34 cases.

(4) Transverse fractures of the petrous pyramid, either pure or combined with longitudinal fractures—16 cases.

All of these cases were further separated into three age groups: (1) those under 20 years of age, (2) those between 20 and 50 years of age, and (3) those older than 50 years of age.

(1) In 49 cases of skull fracture not involving the base there was definite evidence of a perceptive type of deafness in 45 cases. Even after eliminating certain malingerers and some in whom the hearing defect was present before the accident, there were many ears in which the hearing defect was due to the accident. In 4 cases, all under 20 years of age, the hearing was normal; and in general it can be said that the hearing loss was more marked as age increased. In 10 individuals the loss of hearing was of marked degree.

(2) From a study of the cochlear function in cases of unilateral longitudinal fracture one can make several general deductions: A. The function of hearing is more severely damaged by a longitudinal fracture as age increases. B. The hearing loss may be of a conductive, perceptive, or combined type; but the perceptive type of deafness prevails. C. Although the ear on the side of the fracture frequently shows a greater hearing loss than its fellow, still in a large number of cases the loss is almost the same in both ears. In my series, 79.5 per cent of the homolateral ears and 54.6 per cent of the contra-lateral ears showed a considerable hearing loss. This would seem to indicate that the deafness was due to the effects of a concussion of the labyrinth with hemorrhages into the basal coil of the cochlea in both homolateral and contra-lateral ears, rather than to the actual effects of the fracture per se.

(3) A study of the cochlear function in 34 cases of bilateral longitudinal fracture, as evidenced by bleeding from both ears, showed that there was a considerable loss of hearing in 65 per cent of the ears and that in most of these the defect was of the perceptive type. The remarkable feature of this group

was that there were 12 ears with normal hearing and 11 in which there was only slight involvement. This means that in certain cases a fracture may cross the middle ear, tear the drum and cause bleeding from the ear without materially affecting the hearing.

(4) In 16 cases of labyrinthine fracture I found total deafness on the affected side in all the cases. On the contralateral side three ears had normal hearing, three showed a slight involvement, three showed a moderate loss, six showed a marked loss and in one there was an almost complete loss of hearing.

A summary of my investigations of the hearing function after skull fractures of all types would indicate that the hearing was moderately or seriously damaged in 49 per cent of the fractures which did not involve the base. The longitudinal fractures caused a hearing defect in 63.4 per cent of all the ears. The labyrinth fractures caused a complete deafness in all of the homolateral ears, and the hearing was considerably damaged in 65 per cent of the contra-lateral ears. The study would also indicate that the hearing function is more easily and more frequently damaged as age advances. In evaluating these hearing defects one must exercise great care to exclude the deafness due to other causes, particularly in those beyond 40 years of age.

A word should be said about prognosis. The hearing defects found during the first month after a head injury may either improve, remain stationary, or progress. The blood and transudate in the basal coil of the cochlea may slowly absorb, with improvement in the hearing. This is especially true in younger individuals who have not lost much hearing. On the other hand, if the initial loss of hearing has been great, and especially in older individuals, the blood in the cochlear spaces may organize. In this organizing of the blood clot fibrous tissue and later even bone is laid down in the perilymphatic spaces of the cochlea resulting in gradually deteriorating hearing. If such deterioration has not ensued in the first six months it is fair to assume that the condition will remain stationary.

IV.

We are at war. The incidence of head trauma, with its effects upon the ear as we see them in civil life, is going to be greatly increased both in defense workers and in the armed forces. For years the profession has clamored for some method of evaluating in percentage the hearing defects occasioned by trauma. After four years of work, the Committee of Consultants of the Council on Physical Therapy of the American Medical Association presented to the Council a tentative method for evaluating these hearing defects in percentage. This

method was presented to and accepted by the Section of Otolaryngology at the meeting of the American Medical Association last June. It was adopted by the House of Delegates and published in the August 1st number of the *Journal of the American Medical Association.* Until something supersedes it, it becomes the official method of computing hearing losses in percentage. It is not a perfect method and the Committee does not so claim. It is based upon an audiogram taken with an accepted pure tone audiometer. Inasmuch as the hearing and understanding of human speech is the most important consideration from an economic standpoint, the method is designed to give every consideration to those hearing losses occurring in the speech field and little or no consideration to losses occurring outside of this field. Therefore, as the speech field of the audiogram involves the tones from 300 to 4000 cycles, no consideration was given to losses occurring below 256 cycles or above 4096 cycles, and a 10 decibel loss was regarded as threshold. Furthermore, as the middle portion of this speech field has more relative importance than its periphery, the values of each octave were gradually increased from 256 cycles to 2048 cycles and then decreased. As a matter of fact the band of frequencies between 1500 and 2500 cycles represents roughly about 40 to 50 per cent of the important sounds of speech. It was also recognized that the relative importance of a loss increased from threshold up to about a 50 decibel loss on the audiogram and then decreased from that point to the line indicating complete loss of the power to hear and understand speech of a familiar language no matter how loudly spoken. Therefore, the values on the audiogram table were weighted, not only horizontally but also vertically.

We do not contend that this method is perfect. It is not. It is a basic table to cover everyone in general but cannot take into consideration such occupations as music or any other occupation which makes use of the higher frequencies. These are of necessity variables which must be left to courts of law for adjudication. This is true because the method is a method for computing a hearing defect in percentage. It is not in any way a criterion for determining the disability entailed by such percentage loss in the various walks of life. This matter of disability must be left to judicial bodies.

Just how this method will assist in evaluating the disability incurred by members of the armed forces after the war is over is hard to see. Men are being inducted into the service so rapidly that a careful evaluation of their hearing function before induction is impossible. It would be palpably unfair to evaluate their hearing capacity by such a method after demobilization when the only criterion used before induction was the ability to hear numbers in a moderately loud voice at 10 to 20 feet. However, sufficient unto the day is the evil thereof.

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The University of Wisconsin has recently created a Department of Child Development which cuts across college lines and incorporates in one course everything that can be of help to the child that is offered in the various colleges of the University; i. e., medicine, letters and science, home economics, education, etc. At present a student of speech correction can work in the field of speech with special emphasis in speech correction, or in the field of child development with special emphasis in speech correction in which case the student would also be under the supervision of Dr. Robert West who is now also a member of the Department of Child Development.

Dr. Leon Lassers, who received his Ph.D. at Wisconsin in 1942, has recently become Consultant in Speech Correction in the Oregon State Department of Education; he provides a three-way liaison between the public schools, the state department of education and the University of Oregon Medical School.

Miss Mary Fry of Montreal, Canada, who has been studying speech correction at Wisconsin for the last year has become Director of the Speech Clinic at the Children's Memorial Hospital, Speech Pathologist at the Montreal General Hospital and Director of the Speech Clinic at the School for Crippled Children in Montreal. In September she became the wife of Flying Officer Peter Christian Wang of the Royal Canadian Air Force.

Mrs. Emma Hartshorne, who has been the secretary of the speech department at Wisconsin for the past twelve years and is well remembered by many speech correction students, has recently left the University and has become Senior Secretary at West High School, Madison, Wis.

Miss Lorrain Anson, formerly on the staff of the speech department of Purdue University, received her M.A. at Wisconsin last spring and is now research associate at the University of Iowa.

From California come data covering reports of 158 speech correction teachers which show a total of 30,290 students in speech correction classes for the school year 1942-43. Speech handicaps were broadly classified for this report into two groups—nervous speech disorders and articulatory defects (functional and organic). The most common type of speech difficulty among both boys and girls is functional articulatory defects. Stuttering is the next most common type. About three times as many boys as girls exhibit nervous speech disorders, and almost twice as many boys as girls suffer from articulatory defects of speech.

Speech correction in the California schools is carried on by teachers specially trained in speech correction work who have also a background of regular teaching experience. They diagnose the speech defect and provide therapy for the stu-

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PREVENTION AND TREATMENT OF ACOUSTIC TRAUMA[1]

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I.

Acoustic trauma manifests itself by an impairment in hearing. If the trauma has been recent and of brief duration the hearing loss may be temporary, and partial or complete recovery may ensue. If the trauma has been of long duration the hearing loss is permanent. Two different types of hearing loss are observed after acoustic trauma. The most common is the characteristic high tone dip in the audiogram for the tone of 4,096 vibrations, which widens and deepens as the acoustic trauma continues. Incidentally, the first to call attention to this dip as characteristic of occupational deafness was Shambaugh (5) in 1935, confirmed by the observations of Bunch (1) in 1937. The other kind of hearing loss, seen especially after exposure of the ear to a sudden explosive sound, such as a firecracker or sometimes coming from a telephone receiver, consists of a loss of hearing for all tones, with diplacusis. Diplacusis is the symptom where a single tone is heard at a different pitch by the two ears. It appears to be due to a distortion of the vibrating membranes in the inner ear, and where diplacusis follows a sudden very loud sound it is presumably due to the sudden violent agitation of the vibrating membranes, damaging and distorting them.

In both kinds of hearing loss following acoustic trauma there is temporary or permanent damage to the nerve cells in the inner ear. If merely damaged, a nerve cell can recover, but when degeneration and loss of a nerve cell occurs there is no treatment that can improve or restore the hearing, for as far as we know nerve cells cannot regenerate in the human body. Consequently the treatment of acoustic trauma is to recognize the condition as early as possible, prevent further trauma to the ear from this source, and hope that with rest part of the hearing loss may be spontaneously recovered. Most of this recovery will occur within the first few days or weeks after exposure to the traumatizing sound has ceased. We do not know of any vitamin or other medicine which can hasten or further this spontaneous improvement.

Since there is little to be done in the way of treatment for acoustic trauma, prevention is of primary importance. Effective prevention necessitates the early discovery of beginning hearing loss in those whose occupation exposes them to exces-

[1]Read before the American Speech Correction Association, Chicago, December 30, 1942.

sive noise. Since this hearing loss begins for a tone not essential for understanding speech it generally remains unnoticed by the patient in the early stages. Therefore, routine audiometric testing at regular set intervals of all persons exposed to possible acoustic trauma is the only way of discovering the hearing loss before there has been serious damage to the serviceable hearing.

II.

It is clear that the first step in an effective program for the prevention of acoustic trauma is routine audiometric testing of those who are exposed to excessively loud noises.

The second step in a preventive program is the recognition of the fact that some individuals are much more susceptible to acoustic trauma than others. Thus Pastore (4) in a study of the hearing in 88 pilots of commercial airlines found that approximately half of them had normal hearing, although the average flying time of these was four years, and two pilots with 14 years of active flying service had normal hearing. This variation in individual susceptibility to acoustic trauma means that some persons will be able to carry on certain noisy occupations with comparative impunity to their practical hearing, while others, discovered by our routine audiometric tests, must be removed to quieter surroundings before a serious permanent deterioration of their hearing has occurred.

The third step in a program of prevention is the development of effective and practical means of protecting the ears from excessive traumatizing noise. Improved design of machinery, improved factory design to lessen reverberation, and the use of sound-proofing are ideal methods where applicable. The use of sound-proof cabins in airplanes is a step in this direction.

Where the external noise cannot be controlled, as in warfare, the use of an ear protector worn by the individual is of considerable value. It is not necessary that such a protector eliminate all of the excessive noise, nor is it possible to do this. If the noise can be only reduced 20 to 30 decibels the damage to the ears is greatly diminished and may be entirely eliminated. The ideal ear protector should be inexpensive, small, comfortable, easily adjusted and effective.

During the first World War Guild (3) carried out a series of experiments to determine the most effective type of ear protector. He found that a hollow soft rubber bulb with an opening on one side surrounded by a soft rubber flange was effective both in protecting the ear drums against the sudden air pressure changes of a nearby explosion and in diminishing the effect of prolonged exposure to excessively loud sounds. This device was called the "Scientific Ear Drum Protector" or

"Tommy" and was manufactured in Britain toward the end of the last war.

Dickson and Ewing (2) in England recently tested out the efficiency of various types of protectors. These included an ear plug made of metal and rubber, a wax plug covered by cloth that can be molded to fit the ear when softened by body temperature, a tapering closed rubber tube, soft rubber ear pads, and a high altitude flying helmet fitted with earphones as during use in service conditions. Of these various devices the wax plug gave the highest degree of protection and it was as easy to adjust properly as any. By combining the wax plugs with the flying helmet a still higher degree of protection was obtained. These authors emphasize that the intelligibility for speech is diminished in proporton to the protection afforded, and this factor limits the usefulness of any of these devices. For example, aviators who must be able to hear speech through earphones must omit the wax earplugs, though the flying helmet will still afford them partial protection from acoustic trauma. The fact that the hearing for speech is reduced in proportion to the protection afforded very seriously limits the practicability of any form of ear protector, for many persons simply will not use them, or cannot use them and carry out orders properly.

III.

In summarizing the treatment and prevention of acoustic trauma I should like to emphasize the following points and suggest a definite preventive program.

1. Other than the spontaneous recovery which frequently follows a recent acoustic insult as soon as the ear is removed from further exposure, there is no treatment which can restore hearing lost by acoustic trauma.

2. To prevent serious and disabling deterioration of hearing from prolonged exposure to acoustic trauma it is essential that the hearing impairment be discovered at its earliest beginning. This can only be done by routine audiometric testing, for the first loss involves tones not necessary for understanding speech and therefore generally goes unnoticed by the individual.

3. Routine audiometric testing at regular intervals should be instituted in all occupations where the noise is such as to be capable of producing acoustic trauma, in order to find those with more than average susceptibility to acoustic trauma, so that they may be moved to quieter surroundings.

4. Reduction of excessive noise at its source should be encouraged by improved design of engines and machinery, and by improved building design to lessen reverberation.

5. Where the external noise cannot be reduced the wearing of ear protectors by exposed personnel should be encour-

aged, especially if the audiogram reveals the beginning 4,096 dip.

As a closing word let me emphasize once again that the prevention of acoustic trauma requires above all routine and repeated audiometric testing of those exposed to traumatizing noise, for only in this way will the importance of this problem be appreciated by management, labor and the public. What measures should be undertaken by the military to prevent acoustic trauma in aviators, artillerymen, and those exposed to the noise of diesel engines in submarines and tanks, we are hardly in a position to say, but we can urge the need of recognizing this added, and in part preventable, hazard of modern mechanized warfare.

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dent to help him overcome his handicap. Provision has not yet been made to reach all the children in the schools who are in need of remedial speech instruction, but there is an increasing understanding about speech problems on the part of classroom teachers and administrators.

The University of Illinois, Division of Services for Crippled Children, Lawrence J. Linck, Director, is the official state agency established according to the provisions of the Federal Social Security Act for the purpose of extending and improving services for crippled children.

The following types of conditions will be accepted for care: Orthopedic, plastic surgical, neurological, and speech defects associated with cleft palate and cleft lip conditions.

Miss Jayne Shover, formerly State President of the Indiana Speech Correction Association and Director of the East Chicago Speech Correction Program, has been appointed by the Division as Speech Rehabilitation Supervisor for these children under the plan approved by the Children's Bureau. She holds the M.A. degree from Iowa.

Diagnostic speech clinics will be conducted and therapy given at recognized speech clinic centers in public schools, colleges, normal schools, and universities within the state of Illinois.

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HIGH FREQUENCY DEAFNESS AND DISCRIMINATION OF "HIGH FREQUENCY" CONSONANTS*

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I. Introduction

The sounds used in speech long have been defined in terms of portions or bands of the frequency range said to be used in speech. Specific bands of the range have been said to be dominantly important for recognition of and discrimination between specific speech sounds. West says, for example, that all voiced sounds utilize the fundamental frequencies, or the band from 100 to 400 cps.; that the vowels, semi-vowels and nasal consonants utilize the resonance frequencies, or the band from 400 to 2400 cps.; and that the fricative, stop and sibilant consonants utilize the high or friction frequencies, or the band from about 2400 to 8000 cps.[1]

If this theory is true, it should follow that either complete or partial deafness to a given frequency band should affect one's ability in a commensurate degree to recognize and to discriminate between the speech sounds said to utilize that band. For example, if an individual cannot hear sounds in the high frequency range, he should have a corresponding difficulty in recognizing and discriminating the so-called "high frequency sounds" of speech; namely, the stop, fricative and sibilant consonants. Many writers hold this to be true, and contend that defects in the articulation of consonants arise from high frequency deafness, since individuals who cannot discriminate between certain sounds cannot learn to articulate them normally.[2]

*Taken from "Comparison of Auditory Acuity to Pure Tones and the Ability to Discriminate between Sixteen English Consonants," a dissertation submitted to the Graduate Faculty of Louisiana State University in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Speech, by Robert N. Plummer, 1940. This study was directed by Dr. Claude E. Kantner.

[1] Robert West, Lou Kennedy and Anna Carr. *The Rehabilitation of Speech.* New York: Harper and Brothers, 1937, pp. 125-126.

[2] West et al. *The Rehabilitation of Speech*, pp. 125-126.
Alex W. G. Ewing. *Aphasia in Children.* London: Oxford University Press, 1930, pp. 49-74.
Emil Froeschels. *Speech Therapy.* Boston: The Expression Company, 1933, pp. 124-129.
Sara M. Stinchfield and Edna Hill-Young. *Children with Delayed and Defective Speech.* Stanford University, California: Stanford University Press, 1937, pp. 61, 65-74.
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Existence of such a casual relationship between speech development and hearing difficulties in isolated frequency ranges, and between deafness in isolated ranges and the ability to rec-

ognize and discriminate between corresponding speech sounds, is, in the writer's opinion, somewhat in question.

II. Problem

The purpose of the study here reported was to determine the relationship between losses in hearing and consonant discrimination.

III. Procedure

The procedure mainly consisted of six steps:

1. Construction of a speech sound discrimination test involving the eight voiceless consonants [p], [t], [k], [f], [s], [θ], [ʃ] and the affricate [tʃ]; and the eight voiced consonants [b], [d], [g], [v], [z], [ð], [ʒ] and the affricate [dʒ].
2. Administration of the test to 399 Louisiana State University students. The test was administered on all occasions by the writer, orally. Original testing of the 399 was conducted in large classrooms, and to groups of from twenty to thirty. The subjects, facing away from the tester, were seated in a semi-circle. The tester was so located as to be practically the same distance from each subject.
3. Selecting from the group of 399 the 52 subjects whose discrimination test scores made a representative and most normal distribution from poorest to best.
4. Subjecting the 52 to the discrimination test for a second time. This testing was made on groups of from three to five, and conducted in small, semi-sound-proof clinic rooms. Seating arrangement and location of the tester were the same as for previous testing. This second testing was made in order to determine the 52 subjects' discriminatory ability under more favorable conditions than those found in the classrooms.
5. Subjecting the 52 to a test over the pure tones of an audiometer.
6. Making a comparative analysis of the scores made on the discrimination test and the ratings made on the audiometer test.

The Speech Sound Discrimination Test

In testing discriminatory ability between specific sounds of speech, a general sound test will not suffice. A test, for example, which requires interpretation of complete words or sentences is not fine enough to reveal discriminatory difficulty with specific sounds. Words and sentences containing speech sounds which are difficult to perceive may be interpreted upon the basis of meaningful context or perceptible-sound context, or both. In order to determine an individual's ability to recognize or discriminate between sounds of a given frequency band, the sounds said to utilize this band must be presented in relatively isolated form, and not in meaningful context nor in context with other sounds which may give pattern clues.

The speech sound discrimination test best suited to the determination of an individual's ability to recognize the difference between, or similarity of, two speech sounds when presented orally, is a test made up of paired sounds which can be discriminated upon the basis of the least possible number of distinguishing characteristics involved in the sounds of speech. In such a sound test vowels should never be paired with consonants, since two such sounds are said to be distinguishable upon the basis of both friction and resonance frequencies. That is, vowels are said to have resonance but no friction frequencies, and most consonants

have friction but no resonance frequencies. Likewise, voiceless consonants should never be paired with voiced, since they could then be differentiated upon the basis of the presence or absence of the fundamental tone. If, however, we pair vowels with vowels, in theory the only distinguishing differences would lie in their individual resonance frequencies. If we pair voiceless consonants with voiceless, and voiced with voiced, they, in theory, can be distinguished only upon the basis of their differences in the high frequencies. The sound discrimination test used in this study was devised with these principles in mind.

Since this study was concerned only with the high frequency band of the range said to be used in speech, the test utilized only the consonant sounds said to make dominant use of this band. Each pairing in the test made use of the vowel [ə], or of [ə] and [ɑ], to form in every case a pair of nonsense syllables. The test was made up of 312 pairs of such syllables. In 168 pairs the consonants were different ([pɑ-tɑ], [apə-atə], etc.), while in 144 pairs they were the same ([apə-apə], [ap-ap], etc.). In the nature of the pairing it was impossible to have an equal number of "different" and "same" pairs of syllables, but the difference of 24 probably is insignificant. "Same" pairs were included in order that a subject be required to recognize that two consonants were the same approximately as many times as he was required to recognize that two were different.

Each of the sixteen consonants appears in the test 39 times. Each voiceless consonant is paired with every other voiceless, and each voiced with every other voiced, three times, i.e., they are placed initially, medially and finally in nonsense syllables. Each consonant appears 13 times in each position. The position of both consonants in a given pair of syllables is always the same.

Table I shows the sound pairs used. They were given orally in the vertical order shown. As each pair was called out the subject was required to record either an "S" or a "D," according, in his judgment, to whether the consonants in a pair were the same or different. His score was the number of errors. The determined retest reliability of the test was .75± .03.

This test was not standardized. However, the relationship between scores made on it and hearing ability as determined by the audiometer was as follows:

I. Small-group, clinic room discrimination testing (52 subjects):

1. Without exception, 32 subjects making scores in the range 1-9 made audiometer ratings of normal or better.[1]

2. Eight out of 13 making scores of 10-14 made audiometer ratings of normal or better. The remaining five had high frequency losses.

3. Without exception, seven subjects with discrimination scores of 16-122 manifested defective hearing. Four revealed high frequency losses and three severe, general losses.

II. Large-group, classroom discrimination testing (42 subjects):

1. Without exception, 18 subjects making discrimination scores in the range 4-9 made audiometer ratings of normal or better.

2. Out of eight subjects making scores of 12-25, six made audiometer ratings of normal or better, while two had high frequency losses. This score range indicates a three-to-one ratio for normal or better hearing.

3. Of ten subjects making scores of 26-50, five had normal or better audiometer ratings and five had high frequency losses.

4. Six subjects in the discrimination score range of 51 upward rated on the audiometer test as follows: two were normal or better, one revealed high frequency losses and three severe, general losses. This is a two-to-one ratio in favor of defective hearing.

[1] Arbitrarily, plus 10 decibels was set as the line of demarkation between normal and sub-normal hearing ability, and minus 5 as the line between normal and better than normal.

TABLE I. SOUND DISCRIMINATION TEST

pa-ta	θa-θa	ba-ʒa	ba-ba	atʃ-atʃ	aθ-aθ	ag-av	apə-akə	akə-atʃə	abə-dʒə	azə-aðə
pa-pa	ʃa-ʃa	va-va	da-da	ap-ap	aʃ-aʃ	av-av	apə-afə	afə-asə	adə-agə	agə-agə
pa-ka	ka-tʃa	ba-dʒa	za-ða	at-as	aθ-aʃ	ag-az	atə-atə	afə-aθə	azə-azə	avə-avə
pa-fa	fa-sa	da-ga	ga-ga	at-aθ	aθ-atʃ	az-az	akə-akə	atʃə-atʃə	aðə-aðə	azə-azə
ta-ta	fa-θa	za-za	va-va	at-at	aʃ-atʃ	ag-að	apə-asə	afə-aʃə	adə-avə	azə-aʒə
ka-ka	tʃa-tʃa	ða-ða	za-za	ak-ak	atʃ-atʃ	ag-aʒ	apə-aθə	afə-atʃə	aʒə-aʒə	azə-adʒə
pa-sa	fa-ʃa	da-va	za-ʒa	at-aʃ	ab-ad	að-að	apə-aʃə	apə-apə	adʒə-adʒə	aðə-aðə
pa-θa	fa-tʃa	ʒa-ʒa	za-dʒa	at-atʃ	ab-ab	aʒ-aʒ	afə-afə	atə-atə	abə-abə	aʒə-aʒə
pa-ʃa	pa-pa	dʒa-dʒa	ða-ða	ak-af	ab-ag	ag-adʒ	apə-atʃə	asə-aθə	adə-azə	aðə-aʒə
fa-fa	ta-ta	ba-ba	ʒa-ʒa	af-af	ab-av	av-az	atə-akə	akə-akə	adə-aðə	aðə-adʒə
pa-tʃa	sa-θa	da-za	ða-za	ak-as	ad-ad	av-að	asə-asə	afə-afə	adə-adə	aʒə-adʒə
ta-ka	ka-ka	da-ða	ða-dʒa	as-as	ag-ag	adʒ-adʒ	aθə-aθə	asə-asə	agə-agə	adʒə-adʒə
sa-sa	fa-fa	da-da	ʒa-dʒa	ak-aθ	ab-az	av-aʒ	atə-afə	asə-aʃə	adə-aʒə	
θa-θa	sa-sa	ga-ga	dʒa-dʒa	ak-aʃ	ab-að	av-adʒ	aʃə-aʃə	asə-atʃə	adə-adʒə	
ta-fa	sa-ʃa	da-ʒa	ap-at	aθ-aθ	ab-aʒ	ab-ab	atʃa-atʃə	aθə-aθə	agə-avə	
ʃa-ʃa	sa-tʃa	da-dʒa	ap-ap	aʃ-aʃ	av-av	ad-ad	apə-apə	aʃə-aʃə	avə-avə	
tʃa-tʃa	θa-θa	ga-va	ap-ak	ak-atʃ	ab-adʒ	az-að	atə-asə	aθə-aʃə	agə-azə	
pa-pa	ʃa-ʃa	va-va	ap-af	af-as	ad-ag	ag-ag	atə-aθə	aθə-atʃə	azə-azə	
ta-sa	θa-ʃa	ga-za	at-at	af-aθ	az-az	av-av	atə-atə	aʃə-atʃə	agə-aðə	
ta-θa	θa-tʃa	za-za	ak-ak	atʃ-atʃ	að-að	az-az	akə-akə	atʃə-atʃə	agə-aʒə	
ta-ta	ʃa-tʃa	ga-ða	ap-as	af-aʃ	ad-av	az-aʒ	atə-aʃə	abə-adə	aðə-aðə	
ka-ka	tʃa-tʃa	ga-za	ap-aθ	af-atʃ	aʒ-aʒ	az-adʒ	atə-atʃə	abə-abə	aʒə-aʒə	
ta-ʃa	ba-da	ða-ða	ap-aʃ	ap-ap	adʒ-adʒ	að-að	akə-afə	abə-agə	agə-adʒə	
ta-tʃa	ba-ba	ʒa-ʒa	af-af	at-at	ab-ab	aʒ-aʒ	afə-afə	abə-avə	avə-azə	
ka-fa	ba-ga	ga-dʒa	ap-atʃ	as-aθ	ad-az	að-aʒ	akə-asə	adə-adə	avə-aðə	
fa-fa	ba-va	va-za	at-ak	ak-ak	ad-að	að-adʒ	asə-asə	agə-agə	adʒə-adʒə	
ka-sa	da-da	va-ða	as-as	af-af	ad-ad	aʒ-adʒ	akə-aθə	abə-azə	avə-aʒə	
sa-sa	ga-ga	dʒa-dʒa	aθ-aθ	as-as	ag-ag	adʒ-adʒ	akə-aʃə	abə-aðə	avə-adʒə	
ka-θa	ba-za	va-ʒa	at-af	as-aʃ	ad-aʒ	apə-atə	aθə-aθə	abə-aʒə	abə-abə	
ka-ʃa	ba-ða	va-dʒa	aʃ-aʃ	as-atʃ	ad-adʒ	apə-apə	aʃə-aʃə	avə-avə	adə-adə	

The Audiometer Test

The 6B Western Electric audiometer was used to determine the auditory acuity of the 52 selected subjects to the pure tone frequencies said to be used in speech. Each subject was tested at 28 frequencies distributed between 128 and 9747 cps.

IV. Results

Nine of the 52 cases were found to have high frequency losses, ranging from mild to severe. Three were found to have general and rather severe losses over the entire range. Figures 1 through 3 show the better ear audiograms[4] for the high frequency loss group and Figure 4 those of the general loss group. Besides acuity curves, the audiograms show speech sound discrimination test scores (D. S.) and percentages of correct discrimination (C. D.).

Figures 1 through 3 show that these nine cases range from mild to severe in high frequency losses, yet the highest number of discrimination errors out of a possible 312 was 21. This case (Case 49, Figure 1) discriminated correctly 93 per cent of the time. One of the most severe cases (Case 47, Figure 2) discriminated correctly 95 per cent of the time.

The three cases showing general losses present a different picture. If we set somewhat arbitrarily a general loss of 20 decibels or more as the line of demarkation between a relatively mild and a relatively severe one, Case 50 (Figure 4) has mild losses at the frequencies 1024, 1536, 1792, 2048 to 3584, inclusively, and at 5632 and 6655. At all other frequencies his loss is 20 decibels or greater. His hearing is not normal at any frequency, although nearly so at 2460 and 3072. This case was in error 22.8 per cent of the time on the discrimination test.

On the same basis Case 51 (Figure 4) has slightly better than normal hearing at 128 and 512, mild losses at 256 and from 1024 to 1664, inclusive. All other frequencies show severe drops. This case was in error 23.4 per cent of the time. Case 52 (Figure 4), on the other hand, has losses that exceed 20 decibles at every frequency. He was in error 39 per cent of the time on the discrimination test.

On the basis of these three cases it appears that: (1) A relatively general loss is required to produce any considerable percentage of error in discrimination. This is witnessed by the fact that the three cases with general hearing losses (Figure 4) made 71, 73 and 122 errors, respectively, while the highest number of errors among the high frequency loss group was 21 out of a possible 312. (2) Discrimination is chiefly influenced by the amount of loss of hearing in terms of decibels and the extent of the loss on the speech range, particularly the extension of such loss toward the fundamental frequencies. Witness Cases 50 and 51 as against Case 52 (Figure 4). (3) General losses which are comparable on the average, yet quite different in general pattern, may show about the same degree of failure in discrimination. Witness Case 50 as against Case 51 (Figure 4).

[4] The term "better ear" refers to the ear, whether right or left, which had the higher rating on a given frequency.

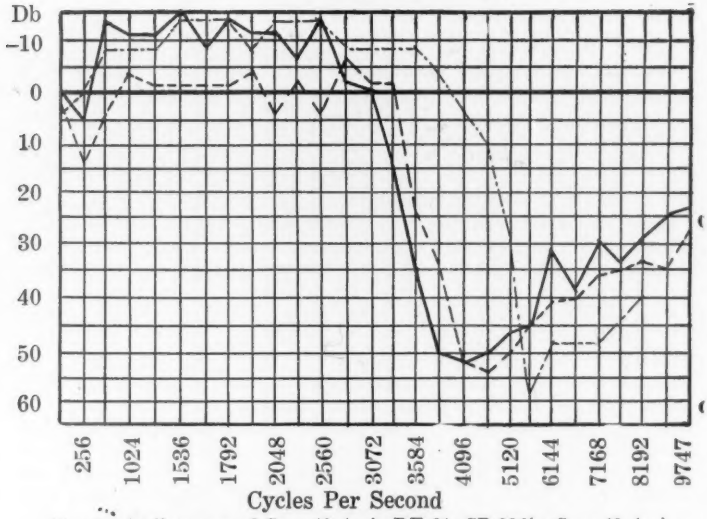

FIG. 1. Audiograms of Case 49 (—), DE 21, CP 93%; Case 48 (- -), DE 17, CP 95%; Case 42 (-.-), DE 12, CP 96%.

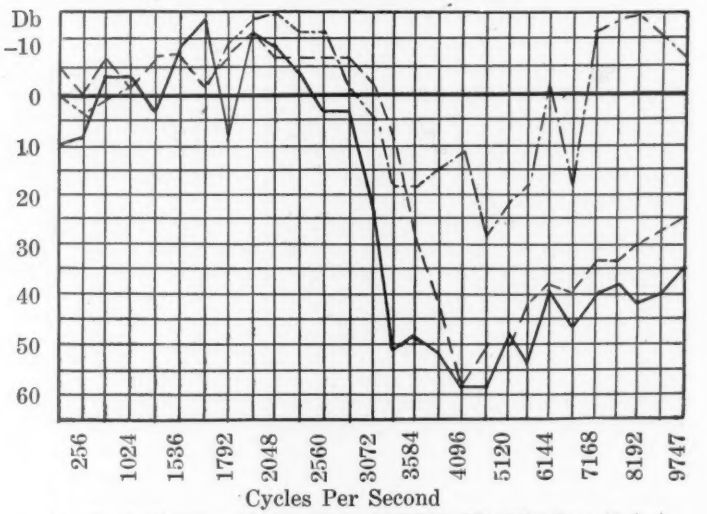

FIG. 2. Audiograms of Case 47 (—), DE16, CP 95%; Case 45 (- -), DE 14, CP 95.5%; Case 46 (-.-), DE 16, CP 95%.

*DE means errors made on discrimination test.
CP means percentage of correct discrimination on the test.

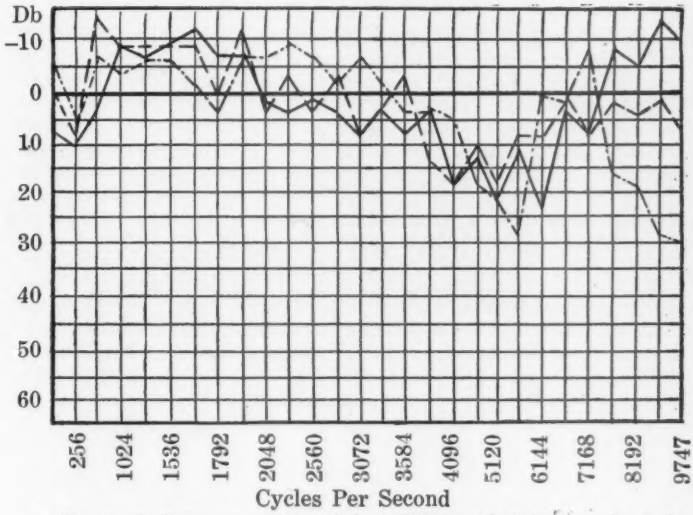

FIG. 3. Audiograms of Case 41 (—), DE 12, CP 96%; Case 34 (- -), DE 10, CP 97%; Case 35 (-.-), DE 10, CP 97%.

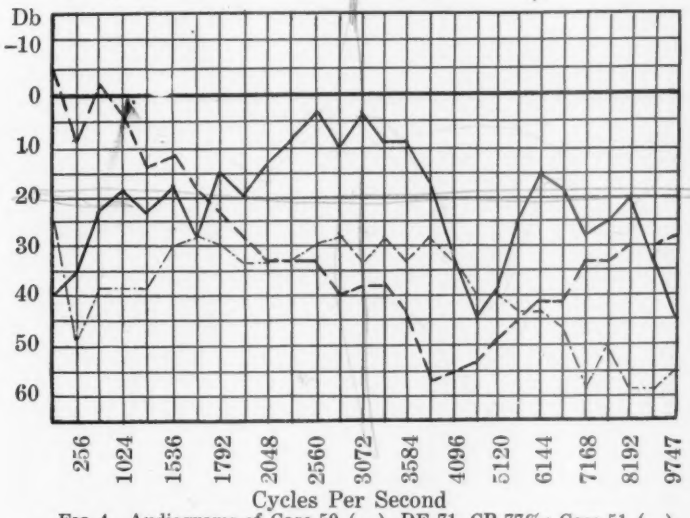

FIG. 4. Audiograms of Case 50 (—), DE 71, CP 77%; Case 51 (- -), DE 73, CP 77%; Case 52 (-.-), DE 122, CP 61%.

*DE means errors made on discrimination test.
CP means percentage of correct discrimination on the test.

Comparison of the discrimination scores of the high frequency and general loss groups with those of the cases having normal or better hearing may be summarized as follows: (1) The nine high frequency loss cases made discrimination scores in the range 10-21. This represents correct discrimination of 93 to 97 per cent for the group. Forty cases with normal or better hearing made scores in the range 1-14, representing correct discrimination of 95.5 to 99.7 per cent. The difference between the correct percentage ranges of the two groups is 2.5 to 2.7 per cent in favor of the normal or better hearing group. (2) On the contrary, the three cases with general hearing losses discriminated correctly only 61 to 77 per cent of the time, as against 95.5 to 99.7 per cent for the normal or better group, representing a difference of 22.7 to 34.5 in favor of the latter. This comparison further tends to show that while general losses have an appreciable influence on ability to discriminate between the consonants, losses in the high frequencies do not.

TABLE II. DISCRIMINATION ERRORS ON [s] AND [z].

Case No.	Point at which losses occur	Severity	[s]	[z]
34	3840-6144	mild*	0	0
35	4608-5632	mild	0	0
41	4096-6144	mild	0	1
42	5120-upward	severe**	0	1
45	3584-upward	severe	2	1
46	3328-5632 and 6656	mild	0	1
47	3328-upward	severe	5	2
48	3584-upward	severe	4	3
49	3584-upward	severe	4	2
50	128-upward	mild to severe, general	3	4
51	1664-upward	severe, general	8	7
52	128-upward	severe, general	10	10

*Generally less than 20 decibels.
**Generally more than 20 decibels.

This study failed to show any relationship between acuity losses at specific frequencies and difficulty in discriminating specific speech sounds. In short, no given consonant appears to be dominantly dependent for its discrimination upon any specific frequency. The most that can be said is that the more severe the general losses, the more prevalently will errors occur among all the sounds. These findings are especially interesting with reference to [s] and [z], two sounds said to be highly dependent upon sensitivity to the friction frequencies. The largest number of errors on these two sounds by any one of the high frequency loss group was seven out of a possible 60 (five on [s], two on [z]). Among the general loss group, 20 errors out of a possible 60 was the highest combined number of errors on the two sounds. A similar study of 40 cases with normal or better hearing reveals an error range of 0-3. Twenty-eight of the 40 made no errors, four erred once each on [s], five once each on [z]. One subject erred twice on [s], another twice on [z] and another once on

[s] and twice on [z]. The errors on the two sounds for the twelve defective-hearing cases reported are shown in Table II. This table also shows the specific hearing losses and their severity.

The general conclusion of this study is that in terms of the percentages of correct discrimination made by the members of the high frequency loss group used in this experiment, the importance of the high frequencies for the fourteen consonants and the two affricates involved has been overemphasized. While it is true that the cases with high frequency losses made slightly more errors than those with normal or better hearing, the increase was extremely small in terms of the possible number of errors on the discrimination test.

This study also creates some doubt as to the existence of a very important causal relationship between articulatory defects involving consonants and high frequency deafness, and suggests that further investigation of this relationship is in order. While it is true that the findings are based upon a relatively small number of high frequency deaf cases, it seems quite significant that, without exception, these cases had no appreciable difficulty in consonant discrimination.

V. Summary and Conclusions

After administering a speech sound discrimination test, composed of the sixteen so-called "high frequency" consonants (including two affricates), to 52 subjects, it was found by audiometer testing that nine of the 52 had mild to severe cases of high frequency deafness. Examination of their discrimination test errors revealed that these nine cases had no appreciable difficulty in discriminating between the consonants which heretofore have been said to depend highly upon sensitivity to the higher frequencies. It also appears that no given consonant sound is dominantly dependent for its discrimination upon any specific frequency along the range said to be used in speech.

Although further investigation is needed before final conclusions can be drawn, the following can be stated tentatively on the basis of the cases reported in the present study:

1. High frequency deafness seems to have no appreciable influence upon an individual's ability to discriminate between the so-called "high frequency" sounds of speech. The term "high frequency" appears to be a misnomer if used to indicate that these sounds depend primarily upon the higher frequencies for their discrimination.
2. The ability to discriminate between these sounds appears to be chiefly influenced by the amount of hearing loss and the extent of the loss along the speech range, particularly the extension of the loss toward the fundamental frequencies.
3. Specific consonant sounds do not appear to be dependent for their discrimination upon specific frequencies in the higher ranges.

ABSTRACTS OF CURRENT ARTICLES

MARY HUBER, *Editor*
University of Wisconsin

Hudgins, C. Visual aids in the correction of speech, *Volta Review*, November, 1935, 637-643.

The stroboscope, an instrument for making voice pitches visible, is used to correct voices of the deaf which are either too high or too low. It also assists in teaching the voice glides characteristic of normal speech.

The pneumodeik is used as a visible test for nasality. There is a small piece of cloth attached to the instrument which moves when the diaphragm inside is moved. One method of using this instrument is to place a small glass bulb in one of the child's nostrils and attach it by means of a rubber tube to the pneumodeik. The flag of the pneumodiek moves when air escapes through the nostril and the child is instructed not to move the flag. A second method is to put a small metal tube in the child's mouth and connect it to the pneumodeik. A lack of flag movement shows a lack of air pressure in the mouth indicating a partially open velum in which case the child is instructed to make the flag move. The pneumodeik may also be used to demonstrate to the deaf the difference in amounts of air pressure required for voiced and unvoiced consonants.

The value of such visual aids in teaching speech to the deaf will depend upon the extent to which there is carry-over into speech situations in which the instruments are not used. *(Abstracted by Lois Silver, Wisconsin.)*

Hughson, W., Ciocco, A., Witting, E. G., and Lawrence, P. S. An analysis of speech characteristics in deafened children with observations on training methods, *Child Development*, 1942, 13, 131-158.

The experiments were performed on 367 children resident in the Pennsylvania School for the Deaf. Of this group, 249 members had never received auricular training up to the time of the study; 118 were enrolled in auricular classes. In general, comparison of the two groups shows that the speech of children given auricular training tends more toward that of the normal child than does that of the oral group. Under all conditions of residual auditory acuity the speech of the children trained by the auricular method is superior to that of the other children. In children trained by the auricular method there is continuous improvement in speech characteristics with duration of training. No comparable improvement is observed for children trained by the oral method. *(Abstracted by Lois Silver, Wisconsin.)*

Mettler, F. A. The present status of auditory research: an anatomophysiologic viewpoint, *J. Gen. Psychol.*, 1940, 22, 387-412.

A detailed description of the anatomy of the human ear is followed by an explanation of the Helmholtz theory, the frequency theory, and a modified frequency theory arising primarily from the work of Wever and Bray. The structure and function of the central auditory pathways is explained which leads to the hypothesis that sounds are analyzed into tones in the medial geniculate body of the thalamus. The view of subcortical functioning is supported by evidence showing that (1) when the temporal cortex is removed deafness does not result, but rather a dysfunction more in the nature of aphasia, and (2) that auditory conditioning is possible after such decortication. Support is given to Henschens' contention that a neurologic spatial arrangement of the auditory system exists which is comparable to that of the visual system. The author's account of the present status of auditory research is particularly well supported by references to recent experimental work in the field of audition. *(Abstracted by Lawrence Norton, Wisconsin.)*

Rayneau, A. J., and Boutet, A. De l'Utilisation du Chloroforme dans la Rééducation des Sourds-Muets Psychiques, *L'Encéphale*, 1916, 9 and 11, 261-270.

A description of a process of achieving improvement in cases of functional deaf mutism through the use of chloroform. The stages through which the patient passes in the course of improvement may include stuttering (especially in the case of former stutterers), monotonous voice, and a residium of partial deafness for a time. Re-educative methods were used in overcoming stuttering. Mutism is regarded as secondary to deafness. A relationship between organic and functional factors is believed to be present in all cases. *(Abstracted by Hide Shohara and Harlan Bloomer, Michigan.)*

Richter, A. Ueber nervoese Hoerstoerungen nach Explosion-swirkung, *Jahrb. f. Psych.*, 1918, Bd. 38, 373.

Three types of functional deafness and deaf mutism are distinguished: (1) Newly acquired lesions (bleeding and perforation) with no symptom of general neurosis. (2) Partly chronic. The deafness is more persistent than the organic conditions indicate; they are not accessible to surprise methods and consciously aggravate their condition. (3) The most serious cases. Show no anatomical injuries; they are extraordinarily apathetic and inhibited; show no reaction to unexpected disturbances; therapy is helpful but brings no change in the serious hearing disturbances. *(Abstracted by Hide Shohara and Harlan Bloomer, Michigan.)*

Selfridge, N. Present status of vitamins in relation to the eighth nerve and conduction deafness, *Arch. Otolaryngol.*, 1941, 34, 125-140.

A valuable contribution is a summary, with special application to the eighth cranial nerve and conduction deafness, of the principle food sources of each vitamin and the obvious signs and symptoms of deficiencies in human beings. "A" deficiency: Xerphthalmia, night blindness, photophobia, difficulty in reading at night, momentary blurring while reading, metaplasias of the conjunctiva and cornea, keratinization in the eye, ear, nose and elsewhere in the body, abscesses in the ear, mucous membranes and elsewhere, keratinization of the skin, and myelin changes in the eighth nerve and other nerves. B complex: "B_1" deficiency: Anorexia, gastrointestinal hypotonicity — constipation or diarrhea — and degeneration of the eighth nerve, spinal cord and other nerves. The B complex is necessary for the metabolism of carbohydrates. "B_2" deficiency: Itching and burning of conjunctiva, photophobia, ciliary congestion, keratitis. The lips are reddened and shiny; maceration and fissuring at the angles of the mouth; seborrhea in the naso-labial folds and scalp; purplish red tongue. "Nicotinic acid" deficiency: Pellagra, with loss of weight, fatigue and gastro-intestinal disorders. The undersurface of the sides of the tongue become fiery red. Symmetric disturbance of the skin, on the face and arms and elsewhere. "B_6" deficiency: Skin lesions—seborrheic and desquamative ulceration of the ears and about the mouth. "C" deficiency: Bleeding from the nose, petechial spots in various parts of the body, swollen and bleeding gums. Experiments have shown changes in middle ear mucosa. "D" deficiency: Rickets. Selfridge believes that deafness is related to all the factors involved in growth—endocrines, electrolytes, amino acids and vitamins. He urges that the time to begin the prevention of deafness is really six months before conception—each parent should be taught that a normal diet is necessary in order to beget a normal healthy child; that such a diet should be continued by the pregnant mother; and that the same careful supervision should be given to the diet of the child, through infancy, childhood, and adolescence.

Shambaugh, G. Heredity in deafness, *Volta Review*, March, 1937, 155-156 and 183-184.

The two types of deafness that can be inherited are congenital deafness and otosclerosis. Congenital deafness may be due to syphilis

in the mother, and it is possible with adequate treatment to prevent transmission of the infection to the child. Congenital deafness may also result from giving quinine to the expectant mother. One-third of the cases of congenital deafness may be said to be inherited and show a family history of deafness. These could be prevented if blood relatives and congenitally deaf persons avoided having children. A further precaution is the avoidance of further pregnancies if normal hearing parents, having congenital deafness in one or both sides of the family, have one child born deaf.

Otosclerosis is an inherited deafness which begins in early or middle adult life. Prevention, rather than cure, must be emphasized, for there is at present no effective treatment for it. Dr. Shambaugh suggests that parents having otosclerosis or persons with a strong family history of deafness avoid having children. *(Abstracted by Lois Silver, Wisconsin.)*

Shambaugh, G. E. and Roberts, W. S. Chronic progressive deafness including otosclerosis and diseases of the inner ear; summaries of bibliographical material available in the field of otolaryngology, *Arch. of Otolaryngology*, 1942, 35, 291-331.

Among the investigations discussed and analyzed by these authors are certain observations of particular interest to speech pathologists, such as (1) the suggestion that hyperparathyroidism may be one of the causes of otosclerosis; (2) the statement that hearing by normal bone conduction is not necessarily via the stapes; (3) that there is, to date, no more satisfactory method of recording hearing acuity and hearing improvement after therapy than the audiogram; that (4) the round window graft operation for deafness seems to be of negligible value; that fenestration of the labyrinth so far is the one surgical technique which appears promising for the alleviation of conduction deafness; (5) Crowe's statement that adenoids recur in more than seventy-five per cent of children whose tonsils and adenoids have been removed before the age of puberty, and that this recurrence often interferes with the ventilating function of the Eustachian tubes and leads to an insidious painless type of progressive deafness; (6) the application of radium in the nasopharynx to reduce lymphoid tissue; (7) the conception of acute and chronic auditory fatigue with the characteristic 4096 dip probably is due to anatomic considerations, the region in the cochlea that responds to 4096 being closest to the oval and round windows and thus is more directly exposed to vibratory energy; (8) Hurst's statement regarding the diagnosis of hysterical deafness in soldiers and civilians and the suggestion that psychotherapy brings more lasting results; (9) the statement that every case of inner ear deafness should have a serologic examination so that cases of syphilitic nerve deafness may be detected early enough to arrest progressive loss of hearing; and (10) Shambaugh's definition of "diplacusis" as the hearing of one sound as two, the three varieties of which are "echo diplacusis," "monaural dysharmonic diplacusis," and "binaural dysharmonic diplacusis."

Thurlow, W. R. Binaural interaction and the perception of pitch, *J. Exper. Psychol.*, 1943, 32, 17-35.

These studies in auditory theory were carried on in the Psychological Laboratory of Princeton University. The results of the experiment indicate that pitch is a function of binaural interaction which produces effects similar to those produced by an increase in the physical intensity. The pitch of a tone sounding in one ear can be changed by introducing a tone of the same frequency in the other ear, provided both tones are of fairly high intensity. The pitch of a tone can be changed by introducing a tone of different frequency in the other ear, provided, again, that both tones are of fairly high intensity. The results of the experiment do not support the hypothesis of the mediation of the perception of pitch by the position of maximal stimulation. *(Abstracted by Lois Silver, Wisconsin.)*

Journal of Speech Disorders, 1943, 8, 385-389

WAR NOTES

HERBERT KOEPP-BAKER, *Editor*
The Pennsylvania State College

The most important recent development in rehabilitation, as it affects the members of the American Speech Correction Association, is that which has occurred in the collateral field of hearing disabilities. Since a large number of speech correctionists have active interest in, and relations with, the rehabilitation of the hard-of-hearing and the deaf, these developments will be reported in these War Notes with some detail:

Certain very direct, powerful, and persistent influences of organized medicine have directed the attention of the rehabilitation officials of the Army, the Veterans' Administration, and the Social Security Agency to the hearing problem. The Army plans to assist deafened soldiers have proceeded far enough to suggest what the general pattern is likely to be in the case of speech disorders. Further, the Army now recognizes officially that the deafened will also require the services of persons specially trained in speech correction. The Office of the Surgeon-General has issued a directive that three Army hospitals are to be designated for the care of hard-of-hearing and deafened servicemen. The hospitals chosen are the Walter Reed Hospital, Washington, D. C.; the Hoff Hospital, Santa Barbara, California; and the Borden General Hospital, Chickasha, Oklahoma. A special ward under Colonel Marion R. Mobley, M.C., has already been set up at the Walter Reed Hospital for soldiers suffering from impaired hearing. Immediate provisions have been made for a capacity of 50 patients. The nursing, medical, and enlisted personnel "for this ward are as nearly permanent as possible in order that they may be trained in the special understanding and the sympathetic care required in the treatment of the deafened." The hospital is also arranging for the part-time services of a psychologist, and each patient is to have certain standardized intelligence and personality tests before admission.

A School of Lip Reading for each patient has been organized providing two or more intensive periods of instruction a day, with added classwork. The courses are to be graded. The civilian staff in the Lip Reading Department will consist of a Supervisor, a number of Lip Reading Aides, and ultimately, several Speech Correction Aides. Two Speech Correction Aides are already on duty in conjunction with this program. The Committee on Rehabilitation of the American Speech Correction Association prepared and submitted recommendations as to qualifications for the Lip Reading and the Speech Correction Aides. This hospital is also to have the services of a Hearing Aid Technician or an acoustic expert, probably a

physicist who will assist the otologist on acoustic problems including the fitting of hearing aids. It is to be equipped with special sound-proof teaching rooms with the necessary electronic equipment for transmitting and amplifying sound either through microphones or from play-back equipment. The use of motion pictures for teaching lip reading is also contemplated. The whole unit is set up with a well-defined research intention.

The Committee on Rehabilitation was in closest touch with Major Walter E. Barton, of the Office of the Surgeon-General, during the preparation of the directive for the establishment of the specialized hearing rehabilitation facilities in Army hospitals. There is good evidence in this directive that the Committee brought an important influence to bear on the policies and procedures as incorporated in the final plans at the Walter Reed Hospital.

The directive from the Office of the Surgeon-General concerning the establishment of services for deafened soldiers clearly indicates that "it is not the intention of the Army to take over the entire rehabilitation program; this is still the province of the Veterans' Administration." There is no evidence, however, that the Veterans' Administration has as yet made comparable plans for the rehabilitation of deafened soldiers who come under its care upon being discharged from the Army. A communication from General Frank T. Hines, Administrator of the Veterans' Administration, insists that that organization "now has facilities in available hospitals to render the necessary treatment to veterans with ear conditions requiring hospitalization." He indicated further that "up to the present time, however, it has not been found necessary to furnish instruction in lip reading as part of hospital treatment in the Veterans' Facilities. This matter is being given attention and at such time as the need arises this treatment will be provided."

The program for the care of veterans with defective hearing who do not require further hospitalization upon discharge from the Army or Navy, and veterans with defective hearing who have received maximum hospitalization and who are discharged from the Veterans' Administration, will be transferred at once to the rehabilitation services of the Federal Security Agency.

Fortunately, however, it has been the attitude of a number of other rehabilitation authorities of the Veterans' Administration that deafened persons *should* be provided with training in lip reading when it can be demonstrated that such training assures improved vocational readjustment and security upon discharge. The Medical Director of the Veterans' Administra-

tion has assured the Coordinator that the counsel of the Rehabilitation Committee of the Association will be sought when the details of physical and educational rehabilitation of deafened veterans are considered.

Early in September the Federal Security Administrator, Paul V. McNutt, announced the establishment of the new Office of Vocational Rehabilitation in the Federal Security Agency to administer the expanded federal-state civilian rehabilitation program provided under the Barden-LaFollette Act of July 6, 1943. The American Speech Correction Association can find considerable satisfaction in the fact that the original Division of Vocational Rehabilitation of the Office of Education has become the nucleus of this new organization. It will be recalled from earlier Notes in the *Journal* that Mr. John A. Kratz was Director of the original Division. Mr. Kratz has been appointed Associate Director of the new Office of Vocational Rehabilitation of the Federal Security Agency. It will be remembered, too, that he is very friendly toward, and deeply interested in, the Association, and has frequently asked for recommendations on speech correction. Recently he has also expressed an interest in the rehabilitation of the deafened, and provisions for this type of service are likely to figure in the program of Federal Security under the non-service-connected legislation.

A late summer communication from Major Walter E. Barton, Medical Corps Assistant of the Office of the Surgeon-General, suggests that the Medical Department of the Army had not yet found much evidence of the need for speech correction in Army hospitals. He writes:

"The Medical Department of the Army thus far is contemplating the use of speech teachers only in connection with the beginning of rehabilitation work with a deaf soldier and in those occasional cases which the special neurosurgical hospital has to treat. This office has requested recommendations and suggestions from the Committee on Rehabilitation of the American Speech Correction Association for inclusion in the program. While there may be a need for speech correction on a larger scale outside of hospital centers, that need has not communicated itself to this office. The screening process has probably eliminated most of the speech cases at the induction point. War casualties have revealed only a very limited number and there has been no request from hospitals for special services. [As you suggest in your letter] rehabilitation work in speech training will, in most instances, be carried out in hospitals under the jurisdiction of the Veterans' Administration."

It is apparent that the medical men of the Army hospitals are not aware of the problem of speech disabilities as a clinical entity and probably do not yet recognize the need of specialized procedures for the treatment of persons suffering from speech disorders.

It is for this reason that the Committee on Rehabilitation

has prepared a circular which describes, in a brief way, the major types of speech and language disorders likely to be found among the war-injured, and the modern clinical methods of dealing with these disorders. This circular is to be sent to all persons directly responsible for the medical care of soldiers. The Coordinator has prepared a special information circular to be sent to the medical staffs of all Veterans' Facilities. There is evidence that these circulars will direct the attention of the medical men, responsible for policies and procedures, to the need for speech rehabilitation more effectively than any other device now available to us.

The Committee on Rehabilitation has now completed the War Service Roster form which is to be sent to the members of the American Speech Correction Association. This Roster will make it possible to provide, more quickly and conveniently, the special speech correction personnel that may be required by the Army, the Veterans' Administration, and the Federal Security Agency.

The Coordinator has visited a number of Army and Veterans' hospitals and has reported his findings in a Memorandum to the Committee on Rehabilitation, dated October 15. The report of his visit to the Valley Forge Hospital at Phoenixville, Pennsylvania, reflects the present status of speech rehabilitation in these centers:

Your Coordinator spent August 25, 1943, in the new Valley Forge Hospital at Phoenixville. This is one of the largest eastern hospitals under Army jurisdiction. Upon describing his mission, he was courteously received by Colonel Smith, Acting Commanding Officer, who immediately established relations with a number of the medical men in charge of Divisions within the hospital.

An interview with Major J. T. Showalter, who is in charge of the Neuropsychiatric Division, revealed that up to the time of the interview, his Division had not received patients requiring specialized speech correction. He felt that any relationship which speech correctionists would have with speech defectives in Army hospitals would not be with neuropsychiatric wards, but rather with Divisions of neurosurgery since all cases suffering war traumas of the peripheral or central nervous systems would be under their care. Any psycho-motor reeducation which these patients received would be directed by the neuro-surgeons. He indicated also that, at least up to the present, the neuro-psychiatrist had tended to conceive of the symptom of stuttering as part of a larger psycho-neurotic picture. He felt that this was not consonant with a modern scientific view of stuttering, and requested information as to how to treat these cases and how to obtain specialized clinicians. The Coordinator also visited the plastic unit of which Colonel J. B. Brown is Chief. Lieutenant Bradford Cannon, his Assistant, was present at the conference. Colonel Brown made it clear that Valley Forge Hospital had received very few head- and neck-injured soldiers up to that time. Of the 52 cases on their records, only one had clear speech implications, which in this case were the result of a destruction of the maxillary bones by shrapnel. The largest portion of the plastic cases received up to that time was for the treatment of severe tank and aircraft burns.

Colonel Brown reported that western Army hospitals were apparently receiving a larger number of head- and neck-injured cases since Pacific combat tended to be characteristically fox-hole fighting in which head and

neck injuries were more frequent. Those cases arriving at eastern Army hospitals tend to have injuries of the arms, legs and trunk. Both Colonel Brown and Lieutenant Cannon recommended that we study the proportion of head and neck injuries in other Army hospitals and make our services available to the plastic men there. They felt confident that the plastic surgeons had a very high regard for the specialized services of the speech correctionists. They insisted, however, that up to that time they had not had need for a person specially trained in speech correction.

A conference with Captain E. R. Mountain, of the Ear, Nose, and Throat Clinic of the same hospital gave the Coordinator evidence that the men in the Head Specialties of the Army hospital were very eager for their deafened patients to receive carefully fitted hearing aids and lip reading training. Captain Mountain was especially interested in the directive from the Office of the Surgeon-General establishing special hospitals for the care of the deafened. He reported that increasingly large numbers of deafened soldiers were being received in his clinic. He asked to be kept informed of the developments at Walter Reed Hospital. Apparently he did not know of the establishment of that unit at the time of our conference.

BOOK REVIEWS

CHARLES VAN RIPER, *Editor*
Western Michigan College

BACKUS, OLLIE L. Speech in Education. New York: Longmans, Green, 1943. Pp. xv + 358.

Speech correctionists have long recognized the need for training classroom teachers in the basic essentials of speech education and rehabilitation, in order that they might more effectively train their students in the adequate use of speech in a general sense, and also cooperate in a program of speech correction in the public schools. In order to advance such a program, the classroom teacher must be afforded the necessary training in speech correction, speech improvement, and in "the uses of speech in everyday living," which Dr. Backus calls "applied speech." *Speech in Education* was written with these purposes in mind.

Following an introductory chapter dealing with the place of speech in education, the book is divided into three main parts, Speech Rehabilitation (11 chapters), Applied Speech (5 chapters), and Speech Improvement (2 chapters). Approximately two-thirds of the material is devoted to speech rehabilitation and the remainder to applied speech and speech improvement. The section on speech rehabilitation contains chapters dealing with the recognition of speech defects, the extent to which the classroom teacher can engage in their diagnosis and correction, and general and special techniques of speech rehabilitation.

The treatment of phonetics is oversimplified to the extent of being erroneous on a few points, and includes the adoption of Kantner and West's innovations in the phonetic representation of diphthongs (e. g., [ej] for [eɪ], [aw] for [aʊ], etc.) The discussion of the physio-anatomical and acoustic aspects of speech appears to be somewhat brief. Since a thorough knowledge of voice science and phonetics seems essential for the diagnosis and treatment of most types of speech defects, a more extended treatment of these topics would have been warranted. The chapters on articulatory and voice defects, foreign accent and hearing problems are well organized and contain practical and usable remedial suggestions. Organic speech disorders are, appropriately enough, treated very briefly, since the classroom teacher cannot be expected to master the technical aspects of diagnosis and treatment of such disorders without obtaining considerably more training than she would have the time or inclination to undertake.

The author's treatment of speech rehabilitation, especially the chapter on stuttering, is based primarily on the readily available texts

of West, Kennedy and Carr and Van Riper, with only cursory reference to a few of the original published researches on which the above texts are based, and with an utter neglect of researches published subsequent to the publication of these texts. This undoubtedly accounts for the somewhat uncritical and non-evaluative treatment by the author of certain aspects of stuttering, as viewed in the light of more recent studies. To cite one example, a statement is made to the effect that "the proportion of certain chemical elements in the blood is found to be different in stutterers as a class from nonstutterers as a class," a statement that does not take into consideration the latest (1940) uncorroborative study of Karlin and Sobel. Furthermore, in her treatment of stuttering, Dr. Backus neglects to distinguish between hypotheses and theories on the one hand, and experimentally derived facts on the other.

The section on applied speech contains chapters on the effective use of speech in education, social life, vocational pursuits and citizenship, with a concluding chapter outlining the necessary teaching procedures. The two chapters in the section on speech improvement outline the criteria of correct speech to be selected by the teacher and present the teaching procedures to be employed. The material in these two sections of the book is both practicable and succinct; the teaching procedures outlined are readily applicable to the classroom situation.

In spite of the above criticisms of various aspects of the material presented, the book as a whole is well written and offers a wealth of useful information on speech education and rehabilitation for the classroom teacher. Dr. Backus should be commended for her realization of the need for a book of this sort and her initiative in writing it. The availability of a book of this nature may serve as a stimulus for the inclusion of courses dealing with speech in education in teacher-training curricula.

EDWARD E. SHULMAN.
University of Iowa

HAHN, EUGENE F. Stuttering: Significant Theories and Therapies, Stanford University, California: Stanford University Press, 1943. Pp. 177. $2.00.

At last we have, within the covers of one book, the concentrated essence of discouragement for beginners in the field of speech correction. Formerly, the neophyte had to wade through masses of scattered literature before coming to the conclusion that, although authorities were very authoritative, the problem of stuttering was largely unsolved. Now, in one clean-cut little book, the graduate student can spare himself library dust and yet "compare and contrast theories of stuttering" on his examinations. Twenty-five (count them) authorities are represented either by their own specially prepared statements of theory and therapy or by Dr. Hahn's competent ghost writing.

In an introductory passage, Dr. Hahn points out the confusion in points of view and dares the reader to find common denominators if he can. An appendix of fourteen pages describes some of the usual procedures in a college speech clinic and urges clinicians to try anything that works.

The main body of the book makes interesting though discouraging reading. These authorities are earnest and sincere and they have all blindly touched the elephant. There is truth in each presentation but it is a partial truth. The attitudes of the authorities are frequently more interesting than their statements. Some are dogmatic; some evangelical; some mystic; but there are no humble authorities.

The theories are evidently far more significant than the therapies, if the space devoted to each is indicatory. Moreover, the compendium certainly makes very clear that although theory is largely etiological, therapy is primarily symptomatic.

What speech correction needs is a Pasteur!

C. V. R.

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STATEMENT OF OWNERSHIP, CIRCULATION, MANAGEMENT, Etc.

Required by the Acts of Congress of August 24, 1912 and March 3, 1933 of the Journal of Speech Disorders, published quarterly at Danville, Illinois for December, 1943. State of Illinois, County of Vermilion, ss. Before me, a notary public in and for the State and County aforesaid, personally appeared Russell L. Guin, who, having been duly sworn according to law, deposes and says that he is the publisher of The Journal of Speech Disorders, and that the following is to the best of his knowledge and belief, a true statement of the ownership, management (and if a daily paper, the circulation), etc., of the aforesaid publication for the date shown in the above caption, required by the Act of August 24, 1912, as amended by the Act of March 3, 1933, embodied in Section 537, Postal Laws and Regulations, printed in the reverse of this form, to-wit: 1. That the names and addresses of the publisher, editor and business manager are: Publisher, The Interstate Printing Co., Danville, Illinois; Editor, Dr. Wendell Johnson, Iowa City, Iowa; Business Manager, Dr. D. W. Morris. Terre Haute, Indiana. 2. That the owner is: (if owned by a corporation, its name and address must be stated and also immediately thereunder the names and addresses of stockholders owning or holding 1 per cent or more of total amount of stock. If not owned by a corporation, the names and addresses of the individual owners must be given. If owned by a firm, company, or other unincorporated firm, its name and address, as well as those of each individual member, must be given): The American Speech Correction Association. 3. That the known bondholders, mortgagees, and other security holders owning or holding 1 per cent or more of total amount of bonds, mortgages, or other securities are: None. 4. That the two paragraphs next above, giving the names of the owners, stockholders, and security holders, if any, contain not only the list of stockholders and security holders as they appear on the books of the company but also, in cases where the stockholders or security holder appears upon the books of the company as trustee or in any other fiduciary relation, the names of the person or corporation for whom such trustee is acting, is given; also that the said two paragraphs contain statements embracing affiant's full knowledge and belief as to the circumstances and conditions under which stockholders and security holders who do not appear upon the books of the company as trustee, hold stock and securities, in a capacity other than that of a bona fide owner; and this affiant has no reason to believe that any other person, association, or corporation has any interest, direct or indirect, in the said stocks, bonds or other securities than as so stated by him. The Intertate, Russell L. Guin, Publisher. Sworn to and subscribed before me this 29th day of November, 1943. Lillian Readnour, Notary Public. (My commission expires January 13, 1945.)